Independent Component Analysis of Personality and Symptoms of Depression and Statistical Parametric Mapping of Personality and Brain Function

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by

Robin Turner

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Abstract

How does personality affect mental illness? Investigations into the Cloninger personality model as a predictor and factor in mental illness have found that significant relationships exist. The relationship between personality and symptoms of depression in a sample of depressed patients before and after treatment is investigated. Utilising the modern brain imaging technique of SPECT the relationship between brain function and personality types in normal males is studied.

Independent component analysis and confirmatory factor analysis are used to investigate new component variables that reduce the data dimensionality and describe response to depression treatment. Two symptom components are found that significantly predict depression outcome. Significant linear and non-linear relationships are found between personality and depression symptoms both before and after treatment using general additive models.

As part of the study, gender differences in personality and symptoms of depression are investigated, using multigroup analysis, leading to a combined symptom structure before treatment. Personality is found to significantly correlate with specific brain regions. In particular the personality trait cooperativeness has significant relationships with brain function in a large number of regions. These results support previous work showing a biological basis for the Cloninger personality model.

Overall the character personality traits appear important in both the relationship with depression symptoms and in the relationship with brain function in normal males. This study has relevance to future randomised clinical trials to assess optimal treatment for depression.

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Papers Resulting from this Thesis

Published Papers

Turner, R., Hudson, I., Butler, P., and Joyce, P. (2003). Brain function and personality in normal males: a SPECT study using statistical parametric mapping. *NeuroImage*, 19:1145-1162.

Conference Presentations

Turner, R. (2000) Personality, Depression and Brain Blood Flow. Christchurch Medical Research Society, Scientific Meeting (New Zealand Medical Journal 2001; 114: 145-146).

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Papers in Preparation

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Turner, R., Hudson, I., Butler, P., and Joyce, P. Confirmatory factor analysis for the latent structure of personality and symptoms of depression. *To be submitted to The British Journal of Mathematical and Statistical Psychology.*

Contents

Chapter 1

Personality, Symptoms of Depression and Brain Function: A Statistical Analysis

Personality has been of interest for many centuries. A wide variety of aspects have been studied, such as, how to adequately describe personality, likewise how to measure personality, what effect personality may have on various psychiatric conditions and what is the biological basis of personality. This thesis takes an indepth look into two aspects of personality.

The first aspect involves the structure of personality in depressed patients, along with the corresponding symptom structure. The changes in personality both pre and post treatment and across gender are probed using a variety of statistical techniques. The second aspect is the investigation of the relationship between brain function and personality types in normal males. Differences in blood flow are studied in relationship to differences in personality types, using relatively new statistical techniques with a novel adaption.

In this study personality is measured using the Temperament and Character Index (TCI) (Cloninger, 1994), symptoms of depression are measured using the Symptom Checklist (SCL-90) (Derogatis, 1983) and brain function, rather than structure, is measured using single photon emission computed tomography (SPECT) (Prohovnik, 1993). Some of the major statistical techniques used in this study include independent components analysis, structural equation modelling and statistical parametric mapping. Personality, depression and brain function are introduced and discussed in the following sections of this chapter.

Chapter 2 details the datasets used in this study. Sections 2.1.1 and 2.1.3 detail the protocols used for data collection. The depressed patient datasets are from the Christchurch Outcome of Depression Study (Joyce et al., 2002) and the Christchurch

Psychotherapy of Depression Study. The second dataset involves the personality types and brain function of twenty normal males. The study protocols, ethical approval and actual study were written, obtained and conducted by the author. Section 2.2 presents some basic descriptive statistics for both datasets including frequency distributions, non-parametric comparisons of medians across groups and nonparametric comparison of the medians pre and post treatment for the depressed patients.

Chapters 3 and 4 present the major investigation of the personality and symptom structure for the depressed patients. The underlying structure of the exploratory data (Christchurch Outcome of Depression Study) is investigated using independent component analysis, which subsumes principal component analysis and factor analysis. Confirmatory factor analysis (CFA) is used to crossvalidate the best structural model on a second dataset from the Christchurch Psychotherapy of Depression Study. These models are used as the basis for further multigroup and longitudinal analyses which investigate gender differences and changes before and after treatment in the personality and symptom structures. Discriminant analysis and logistic regression are used to interpret the factors from the confirmatory factor analysis.

The prediction of symptoms by the personality variables, and vice versa, using path analysis with latent variables and general additive models is presented in Chapter 5. This chapter investigates how much can be known about a patient's personality by their depression symptoms and vice versa. The general additive model allows for non-linear modelling of any potential relationship.

Chapter 6 presents the analysis to test for any relationship between brain function and personality using statistical parametric mapping (SPM). A number of preliminary steps are needed before the SPM can be implemented. A novel approach was taken to group the personality predictors into quartiles and modelling was achieved using a general linear model with contrasts between the various quartile levels (assessed for significance) and regional cerebral blood flow. The results of Chapter 6 are published in Turner et al. (2003). Chapter 7 summarises the results presented and suggests avenues for future work.

1.1 The Quantification of Personality, Symptoms of Depression and Brain Function

Reliable measurement of personality, symptoms and brain function are required for sound statistical investigation. Over the years questionnaires and methodologies have been developed to quantify the personality and symptom variables. This chapter discusses the specific tools used in this study to measure personality, symptoms of depression and brain function in light of recent developments in these fields.

A variety of personality models are in use today, each with their own advantages and disadvantages. A recently developed model that offers the advantage of a biological basis is the Temperament and Character Inventory (TCI) developed by Cloninger et al. (1994). This model introduces seven basic traits that measure unique personality axes. These traits are novelty seeking (NS), harm avoidance (HA), reward dependence (RD), persistence (P), self directedness (S), cooperativeness (C) and self transcendence (ST). The amount of these traits, and the interaction between them, describe how a person behaves and reacts to situations, and can distinguish between certain psychiatric disorders (Pervin, 1993). Section 1.2 discusses the TCI model, introducing the rationale for and the importance of its use.

The Symptom Checklist (SCL) (Derogatis, 1983) has been used in this study to measure the psychological distress of a person at a particular time. The questionnaire contains 90 questions which relate to nine symptoms, namely somatisation (S), obsessive-compulsive (OC), interpersonal sensitivity (IS), depression (D), anxiety (A), anger-hostility (AH), phobic anxiety (PA), paranoid ideation (PI), and psychotocism (P). There is considerable debate as to the number of factors this questionnaire actually measures (Vassend and Skrondal, 1999). The main area of contention is whether the SCL questionnaire measures an overall factor of distress, or a multitude of symptoms. Section 1.3.1 introduces and discusses the SCL questionnaire.

Single Photon Emission Computed Tomography (SPECT) (Prohovnik, 1993) is used in this study to measure brain function. Brain function is the activity or working of the brain rather than the physical structure of the brain. Brain function can be measured in a variety of ways such as the electromagnetic activity in the brain, the amount of oxygen use in parts of the brain or the amount of blood flow, which is highly related to oxygen uptake. In this technique a radioactive isotope is injected into the blood stream and is taken up by the brain in direct proportion to blood flow. Thus levels of the radioactive tracer indicate functional activity in the brain. Gamma cameras are used to detect radioactive decays from the tracer. The cameras take two-dimensional pictures of the brain, which are then reconstructed to a three-dimensional picture using tomography (Hounsfield, 1973). The three dimensional image is a map of the counts per pixel of radiation in the brain (Knoll, 1983). As the tracer is taken up in proportion to blood flow, high areas of activity indicate high blood flow. This in turn indicates increased brain function in the region. The technique of SPECT was developed from a combination of developments with radioisotopes and the advent of computerised tomography (Larsson and Israelsson, 1982). These developments and further theory behind SPECT imaging are discussed in Section 1.5.

1.2 Personality

Personality is defined by Pervin (1993) as

> Those characteristics of the person that account for consistent patterns of behaviour.

The theories developed in the *20th* century to describe personality fall into a number of categories including psychoanalytic theory, phenomenological theory, cognitive theory and trait theory as detailed below.

The following section uses the definitions as described in Pervin (1993). The psychodynamic approach to the study of personality uses the interaction of motives, drives, needs and conflicts and was introduced by Freud (Freud, 1933, 1940, 1953). The phenomenological approach, developed by Rogers (1947, 1961, 1964), is based on an individual's perceptions of themselves and their surrounding world and has "fulfilment of potential" as the driving force. Cognitive theory uses constructs to describe personality. These constructs are unique to the individual and are developed from experience and then used to predict and interpret events (Kelly, 1955). Trait theory, developed and refined by Allport (1937); Allport and Allport (1921); Eysenck (1947, 1970); Cattell (1956), bases a person's behaviour on a set of traits. The traits are the same across people, but individuals vary on the levels of each trait. Trait theory suggests individuals will have a predisposition to behave in a certain way according to their trait levels. Traits are often developed using factor analysis. There are other types of descriptive systems for personality, further information can be found in Pervin (1993).

This study uses a trait based model (Temperament and Character Inventory) as it allows for a quantification of personality across individuals. A commonly used trait model is the Five-Factor Model, often called the Big Five (Costa and McCrae, 1985). The five traits measured are Neuroticism, Extraversion, Openness, Agreeableness and Conscientiousness. A recent development in trait theory is the Cloninger model of personality (Cloninger, 1994). This study uses the Cloninger model, which is discussed in detail in the following section.

1.2.1 The Temperament and Character Inventory

Cloninger developed the Temperament and Character Inventory (Cloninger, 1987). The index has seven basic axes, or traits, that are categorised into either temperament or character. The temperament descriptors (novelty seeking, harm avoidance, reward dependence, persistence) are moderately heritable and stable (Cloninger et al., 1994). This suggests an individual is born with particular temperament traits, which remain similar through out life. Cloninger et al. (1994) describes temperament as the "automatic

emotional responses to experiences". Character (self directedness, cooperativeness, self transcendence) on the other hand is described by Cloninger et al. (1994) as referring to the "self concepts and individual differences in goals and values that influence voluntary choices, intentions and the meaning of what is experienced in life". Thus, unlike temperament, character changes with age and is influenced by social experiences.

The traits are measured by a self report questionnaire (Cloninger et al., 1994). The 240 question version of the Temperament and Character Index has been used in this study. Each question relates to a single trait. Initial analysis of the questionnaire calculates 25 subscales, which are presented in Table 1.1. The traits are shown in the first column and the subscales for each trait listed with a description (from Cloninger et al. (1994)). The subscale RD2, was originally part of reward dependence, but later studies presented evidence for it being a trait in its own right (Cloninger et al., 1993). Table 1.2 presents the descriptors for high or low scores on each of the temperament traits and Table 1.3 for the character traits. The descriptions are from Cloninger et al. (1994).

The resulting data from the TCI model are based upon a summation of the scores for each question; in the bivariate form of the self report form a zero/one coding system is used. Calculation of the TCI subscales is a straight forward mean response for the questions relating to a particular trait. Missing data are accounted for by calculating the mean across only those questions actually answered at an individual level. The levels of the scales within and between individuals are then directly comparable. To calculate the seven TCI traits from the subscales, a mean of the subscales is calculated.

1.3 Depression

Joyce (1995) summarises the criteria for a major depressive epsiode (DSM-IV, 1994) as a period of at least two weeks within which there is either a depressed mood or a loss of interest or pleasure in most activities. This must be accompanied by at least four additional symptoms from a list that includes changes in appetite or weight; disturbed sleep; altered psychomotor activity; decreased energy; feelings of worthlessness or guilt; impaired concentration, thinking or decisiveness; and recurrent thoughts of death or suicidal ideation or behaviour. These symptoms must be present most of the day, nearly every day, for at least two weeks, and be associated with significant distress or impairment. This impairment may include apparently normal functioning, but the individual requires markedly increased effort to carry on with normal activites, often the individual continues to work but ceases most personal and family activities.

To measure the severity of the depression symptoms, two main groups of scales are used. The first group are the clinician rated scales such as the Hamilton depression rating scale. The second group are the self report scales such as the Symptom Checklist. This

Trait	Definition	Subscale	Definition/Description
NS	Novelty Seeking	NS1	Exploratory Excitability vs Stoic Rigidity
		NS2	Impulsiveness vs Reflection
		NS3	Extravagance vs Reserve
		NS4	Disorderliness vs Regimentation
HA	Harm Avoidance	HA1	Anticipatory Worry vs Uninhibited Optimism
		HA2	Fear of Uncertainty vs Confidence
		HA3	Shyness with Strangers vs Gregariousness
		HA4	Fatigability & Asthenia vs Vigor
RD	Reward Dependence	RD1	Sentimentality vs Insensitivity
		RD3	Attachment vs Detachment
		RD4	Dependence vs Independence
P	Persistence	RD2	Persistence vs Irresoluteness
S	Self Directedness	S1	Responsibility vs Blaming
		S2	Purposefulness vs Lack of Goal Direction
		S3	Resourcefulness
		S4	Self-Acceptance vs Self Striving
		S5	Congruent Second Nature
C	Cooperativeness	C1	Social Acceptance vs Social Intolerance
		C2	Empathy vs Social Disinterest
		C3	Helpfulness vs Unhelpfulness
		C4	Compassion vs Revengefulness
		C5	Integrated Conscience
ST	Self Transcendence	ST1	Self-Forgetfulness vs Self-Conscious Experience
		ST2	Transpersonal Identification vs Self-Isolation
		ST3	Spiritual Acceptance vs Rational Materialism

Table 1.1: The Temperament and Character Index traits, 25 subscales and descriptions.

Temperament Traits	High Scorers	Low Scorers
Novelty Seeking	exploratory & curious	indifferent
	impulsive	reflective
	extravagant & enthusiastic	frugal & detached
	disorderly	orderly & regimented
Harm Avoidance	worrying & pessimistic	relaxed & optimistic
	fearful & doubtful	bold & confident
	shy	outgoing
	fatigable	vigorous
Reward Dependence	sentimental & warm	practical & cold
	dedicated & attached	withdrawn & detached
	dependent	independent
Persistence	industrious & diligent	inactive & indolent
	hard-working	gives up easily
	ambitious & overachiever	modest & underachiever
	perseverant & perfectionist	quitting & pragmatist

Table 1.2: Descriptors of the temperament traits.

Character Traits	High Scorers	Low Scorers
Self-Directedness	mature & strong	immature & fragile
	responsible & reliable	blaming & unreliable
	purposeful	purposelessness
	resourceful & effective	inert & ineffective
	self-accepted	self-striving
	habits congruent with long term goals	habits incongruent with long term goals
Cooperativeness	socially tolerant	socially intolerant
	empathic	critical
	helpful	unhelpful
	compassionate & constructive	revengeful & destructive
	ethical & principled	opportunistic
Self-Transcendence	wise & patient	impatient
	creative & self-forgetful	unimaginative & self-conscious
	united with universe	pride & lack of humility

Table 1.3: Descriptors of the character traits.

SCL-90-R General Symptoms	Example symptoms
Somatisation	Pain in body, faintness, nausea.
Obsessive Compulsive	Repetition of actions, double checking
Interpersonal Sensitivity	Hurt easily, critical of others, self conscious
Depression	Low energy, self blaming, blue
Anxiety	Nervousness, tense, scared
Anger Hostility	Easily irritated, feeling violent, shouting
Phobic Anxiety	Fear of open spaces, fear of crowds, fear of being alone
Paranoid Ideation	Blaming others, feeling of being watched, not trusting people
Psychoticism	Hearing voices, idea that something is wrong with body or mind

Table 1.4: Symptoms measured by the SCL-90-R.

study focuses mainly on the Symptom Checklist, however in Chapter 4 the Hamilton depression rating scale is used as a single measure of depression severity and thus an indicator of improvement when veiwed across time.

1.3.1 The Symptom Checklist

The Symptom Checklist (SCL-90-R) was developed by Derogatis (1983) to measure symptoms of psychological distress. These symptoms are measured using a 90 question self report form. The nine symptoms are Somatisation (S), Obsessive Compulsive (OC), Interpersonal Sensitivity (IS), Depression (D), Anxiety (A), Anger Hostility (AH), Phobic Anxiety (PA), Paranoid Ideation (PI) and Psychoticism (P). Table 1.4 presents examples of some of the symptoms experienced for each of the nine general symptoms.

There is debate as to how many components the SCL questionnaire actually measures. Some studies, such as those by Carpenter and Hittner (1995), Bonynge (1993) and Bernstein et al. (1994), have shown evidence of a single overall factor of general distress, rather than nine distinct symptoms as presented by Derogatis and Cleary (1977). However, Bernstein et al. (1994) suggested a second factor may be appropriate, separate from the overall distress measure. Steer et al. (1994) not only found an overall general component of distress, but also identified four specific residual components that were appropriate for their study. Studies such as those by Vassend and Skrondal (1999) and Schwarzwald et al. (1991) also presented evidence for more than one factor.

A second point of contention with the symptom checklist is to the presence or absence of gender differences in the symptom structure. Gender differences are investigated in this thesis. Bonynge (1993) showed gender invariance in a group of suicidal adults and adolescents. Vassend and Skrondal (1999) and Carpenter and Hittner (1995) both showed significant gender differences, on data from, in the first case, the general population and data from psychiatric patients. These conflicting results may be partly due to the types of

individuals analysed (for example differences in illness type, stage of illness, or treatment type).

1.3.2 The Hamilton Depression Rating Scale

The Hamilton Depression Rating Scale (Hamilton, 1960, 1967) is a seventeen item list of symptoms that are measured for severity by a clinician rather than self reported. The scale is used to score the results of an interview and the seventeen items measure aspects such as depressed mood, guilt, suicide, insomnia, anxiety and somatic symptoms. The scale was developed to give information about depression symptoms and be closely related to diagnosis specific to depression rather than mental illness in general.

1.4 The Relationship between Personality and Symptoms of Depression

The early studies into the relationship between personality and depression found that the personality characteristics of neuroticism and extraversion showed some relationship with depression (Frank et al. (1987), Hirschfeld and Klerman (1979), Hirschfeld et al. (1983a), Hirschfeld et al. (1983b), Hirschfeld et al. (1989), Kerr et al. (1970), Liebowitz et al. (1979) and Weissman et al. (1978)). Generally the studies show that higher neuroticism scores are found in depressed patients than controls and neuroticism scores decrease slowly with improvement of the depression. Low extraversion generally related to more severe depression. Personality models have been developed that measure the personality characteristics most associated with depression (Akiskal and Hirschfeld, 1983; Beck et al., 1983). The relationships between these characteristics and depression have been studied, for example by Hirschfeld et al. (1976), Hirschfeld et al. (1977), Hirschfeld et al. (1986), Rohde et al. (1990), Boyce and Parker (1989), Blatt (1974) and Frank et al. (1987).

During the 1990s the Cloninger model has been investigated in relation to depression. Joffe et al. (1993) investigated novelty seeking, harm avoidance and reward dependence before and after treatment in 40 unipolar depressed patients. They found harm avoidance had significantly higher values in non-responders than responders (to treatment).

Mulder and Joyce (1994) showed that harm avoidance was significantly correlated with the level of depression. Joyce et al. (1994b) investigated the depression outcomes in relation to personality in 84 depressed patients. Rather than model just the temperament traits directly to the percentage improvement in depression, they dichotomised the three variables, novelty seeking, harm avoidance and reward dependence, into high and low, to give eight temperament types. Using multiple regression techniques they found that these eight types accounted for 25% of the variance in treatment outcomes.

Joyce et al. (1994a) used a sample of 94 depressed patients and 40 normal controls to investigate daily fluctuations in cortisol levels. Hypersecretion of cortisol is often found in depressed patients. They found that reward dependence was significantly correlated to cortisol level. Nelson and Cloninger (1995) found that harm avoidance was correlated to the pre-treatment depression severity, as measured by the Hamilton Depression Rating, and reward dependence was correlated to the change in Hamilton Depression Rating during treatment.

The most recent studies investigate all seven of the Cloninger traits in relation to depression. Farmer et al. (2003) investigated the genetic vulnerability to develop depression and found that novelty seeking, harm avoidance, reward dependence and self directedness were related to depression. In another study (Agosti and McGrath, 2002) depressed patients were treated with fluoxetine, imipramine or placebo. Personality was measured before treatment and then 8 weeks later. The study found that those who responded to treatment had significantly reduced harm avoidance but still higher than the normal controls, and self directedness levels returned to normal. The two different drugs did not result in differences in personality traits except self transcendence.

Luty et al. (2002) showed strong correlations between interpersonal sensitivity and temperament and character. Marijnissen et al. (2002) found that harm avoidance scores were significantly higher in depressed patients before and after treatment compared to normals. In their study the TCI scores were not predictive of response to treatment. In a study of 108 depressed patients (Hirano et al., 2002) the Hamilton depression rating was positively correlated to harm avoidance and negatively correlated to self directedness and cooperativeness. In the responders group these scores improved with symptom improvement. Dysfunctional attitudes in depressed patients were related to self directedness in a study by Luty et al. (1999).

Corruble et al. (2002) investigated the personality changes in patients recovering from depression. They found that early changes involved decreased harm avoidance, and increased self directedness and cooperativeness. Naito et al. (2000) showed that self directedness could be predictive of depression. Sato et al. (1999) used logistic regression to predict drug response from TCI personality scores. They found that the character traits self directedness and cooperativeness were important. Hansenne et al. (1999) found that self directedness and cooperativeness were related to depression severity using the Hamilton scale.

The most recent study, as far as the author is aware, to investigate the Cloninger temperament model in relation to depression is presented in Grucza et al. (2003). Grucza et al. (2003) investigated the relationship between the temperament dimensions (novelty seeking, harm avoidance, reward dependence and persistence) and depressive symptoms measured by the Center for Epidemiologic Studies Depression Scale (Radloff, 1977) in a

sample of 804 adults from the general population. The study used canonical correlation analysis and logistic regression. The first personality canonical correlation component measured mainly harm avoidance and was positively correlated with a symptom component measuring an overall severity of symptoms. The second personality component constituting reward dependence and persistence was positively correlated with a symptom component that mainly measured vegetative symptoms and dysphoria. Logistic regression was used to investigate other measures of depression in relation to the temperament measures.

The study by Grucza et al. (2003) concentrated on the temperament personality variables. Likewise most of the studies using the Cloninger personality model have concentrated on the temperament variables (Enns and Cox, 1997).

1.5 Measuring Brain Function

A variety of modalities have been developed to investigate brain function as opposed to brain structure. These include functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET) and Single Photon Emission Computed Tomography (SPECT). PET and fMRI are unavailable in Christchurch thus the study uses SPECT imaging.

SPECT uses the measurement of regional cerebral blood flow (rCBF) as an indicator of brain function. The two are well correlated (Meyer, 1978). The process starts with the injection of a radioactive isotope that gets taken into the brain in proportion to the blood flow. The radioisotope emits gamma rays that are detected by gamma cameras. Tomographic reconstruction calculates the three dimensional distribution of the radioisotope, giving an image of the blood flow in the brain at the time of isotope injection.

A variety of brain functions have been investigated with brain imaging techniques such as SPECT and PET. For example, responses in the normal brain to stimuli such as light, noise and pain have been investigated (Phelps et al., 1981; Mazziotta et al., 1982, 1984; Buchsbaum et al., 1983). Extensive investigations into cognitive activation have also been published. Some of these cognitive functions include concentration, attention and apprehension (Meyer, 1978), and the difference between resting and a psycho physiologic task (Meyer et al., 1980). Drevous (1989) discusses the historical use of SPECT imaging in the psychiatric field, where various disorders have been investigated including affective disorders (Baxter et al., 1985), schizophrenia (Weinberger et al., 1986) and dementia (Rogers et al., 1986). The following section presents the details involved in SPECT imaging.

1.5.1 SPECT

SPECT imaging begins with the injection of a radioactive isotope that is attached to a chemical which has the appropriate properties to get to the site of interest. The radioactive tracer of choice for SPECT brain imaging is 99^m Technetium Hexamethylpropylene Amine Oxime (99^mTc HMPAO). The radioactive isotope is 99^m Technetium (commonly used in medicine and available in most nuclear medicine departments) which is a gamma ray emitter with a short half life. HMPAO was developed to have properties of long retention time and stability in the brain, good distinction between grey and white matter, ability to pass through the blood brain barrier, low toxicity and reasonable dosimetry. The maximum brain uptake of the injected dose is 5%. Within two minutes only 15% of this is returned to the blood stream (Amersham, 1994). The distribution in the brain remains stable and the radiation levels drop following the isotope half life (Amersham, 1994).

Initial work with HMPAO[1] demonstrated it had the appropriate properties for functional brain imaging and investigated the kinetics of tracer uptake post injection. Some preliminary studies into the use of SPECT HMPAO in stroke, epilepsy, extrapyramidal disorders, dementia, headaches, psychiatric disorders, and neuropsychological studies were published by Podreka et al. (1987). Matsuda et al. (1992) demonstrated the difficulties of achieving quantitative SPECT HMPAO because of the rapid conversion between diffusible and non-diffusible 99^mTc-HMPAO in the brain and blood stream. Thus most SPECT studies measure counts of radiation rather than the actual blood flow as such.

Figure 1.1 shows the SPECT imaging and reconstruction technique. This study used a GE systems scanner and software for the data collection and tomographic reconstruction. The scanner had a dual head rotating gamma camera that moved through a 180° arc, taking images in a step and shoot series. This resulted in 64 two dimensional projections. These two dimensional projections were then reconstructed by firstly filtering to reduce noise[2], then transforming to the Fourier space[3] and filtering againto recondition the data. After the inverse Fourier transform is used, the filtered data is back projected to build up a three dimensional image of the original distribution of radiation, which is in proportion to blood flow at the time of injection and thus an indicator of brain function (Knoll, 1983).

[1]The following papers presented results from various investigations of the HMPAO properties and kinetics; Nowotnik et al. (1985); Holmes et al. (1985); Costa et al. (1986); Andersen et al. (1987); Neirinckx et al. (1988); Yonekura et al. (1988); Lassen et al. (1988); Andersen (1989).

[2]The noise comes from scattered and background radiation and usually contributes to the high frequency components so smoothing or filtering in Fourier space can reduce the noise.

[3]Fourier space is the frequency domain for the images calculated using Fourier transforms.

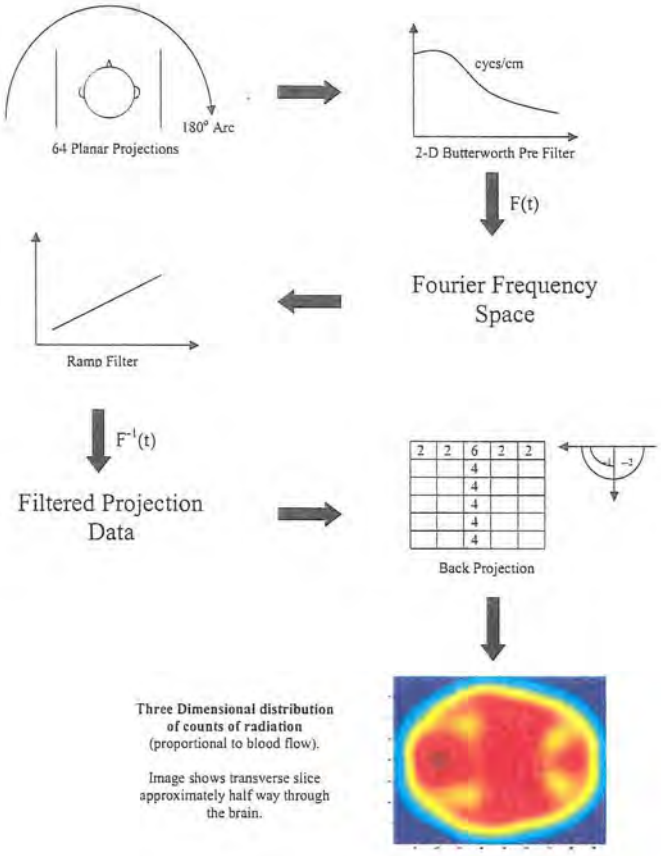


Figure 1.1: The back projection reconstruction technique.

1.6 Brain Imaging and Personality

Fischer et al. (1997) used PET to investigate the relationship between extraversion, neuroticism (traits from the NEO personality model) and regional cerebral blood flow. The study found that extraversion appeared to be related to the subcortical (caudate nucleus, putamen) brain regions. The study was unable to find any differences in rCBF related to neuroticism. Johnson et al. (1999) also used PET to investigate rCBF in relationship to introversion/extraversion. They found introversion was related to increased rCBF in the anterior thalamus and frontal lobes, where as extraversion related to rCBF in the anterior cingulate gyrus, the temporal lobes and the posterior thalamus.

The relationship between three of the Cloninger TCI traits and rCBF was investigated in normal volunteers by Sugiura et al. (2000) using SPECT. This study found significant correlations between all three TCI traits (NS, HA, RD) and rCBF. NS was positively correlated with rCBF in the paralimbic regions and both HA and RD were negatively correlated with various neocortical regions as well as the paralimbic cortex.

Moresco et al. (2002) used PET to investigate receptor binding of serotonin $5HT_{2A}$ in relationship to NS, HA and RD. They found HA was negatively correlated with serotonin binding in the cerebral cortex. Vedeniapin et al. (2001) showed that the P300 event-related brain potential was significantly reduced in low scorers on the self directedness TCI trait. More details on the status of functional brain research related to personality can be found in Cloninger (2002).

Viinamaki et al. (1998) used SPECT imaging to investigate monoamine transporter density in personality disorder. The study showed lower monoamine uptake, compared to normal controls, in the medial prefrontal area and thalamus. After treatment, uptake was normal whilst in the control patient the levels remained low. Multiple personality disorder was investigated with SPECT by Saxe et al. (1992). Scans were taken when the patient was in different personality states. The study suggested that the temporal lobe was involved in the psychophysiology of multiple personality disorder. The study by Menza et al. (1995) suggested that personality traits in Parkinsons disease patients may be affected by striatal deficits in dopamine. Specifically novelty seeking was related to tracer uptake in the left caudate.

This thesis investigates personality in two areas. The first area investigates the structure of personality and symptoms of depression before and after treatment including gender differences and changes across time. The relationship between personality and symptoms of depression are also investigated. The second part of the personality study investigates the relationship between regional cerebral blood flow and personality types in normal males. The following chapter introduces the datasets that are analysed, giving details of study protocols and descriptive statistics, provides a general overview of the

features of the data and tests for possible gender and time effects. How "normal" are the so-called "normals", in the brain study, is also briefly investigated.

Chapter 2

The Measurement of Personality, Symptoms of Depression and Brain Function

The previous chapter introduced the methodologies of measuring personality, symptoms of depression and brain function. This chapter delineates the datasets that were used in the study and presents some descriptive statistics of the data. There are two distinct themes to this thesis. The first is an investigation of the personality and symptoms of depressed patients including the change in these after treatment. Two datasets are used for these investigations. The first will be used as an exploratory dataset used for model development and the second as a validation dataset. Both datasets are from the Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences (Carter et al., 2000; Luty et al., 1999; Mulder et al., 1999) and contain information on the personality and symptoms of depressed patients pre and post treatment. The second theme investigated in this thesis, is the relationship between personality and brain function. Data was collected, by the author, from twenty normal volunteers who completed the TCI questionnaire and underwent SPECT imaging.

To complement this work a brief investigation was made into how normal the volunteer's personality actually was. The Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, has data collected from the never ill relatives of bipolar patients giving a "normal" data set that will be used for the comparison. Bipolar disorder involves cycling between depressed and manic phases. Research into the hereditability of bipolar disorder has shown genetic links (Sevy et al., 1995) indicating that using the family members of bipolar patients as a normal dataset will have some bias depending on the relationship between the genetic bipolar traits and any associated personality traits.

Sections 2.1.1 to 2.1.4 present the details used for data collection by the Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences and the author. The depressed patient data and the data from the never ill relatives of bipolar patients is from the Department of Psychological Medicine. The data for the brain study was collected by the author with the help of the Nuclear Medicine Department, Christchurch Hospital and was funded by the Department of Psychological Medicine and Nycomed New Zealand Limited.

2.1 The Datasets

2.1.1 The Depressed Patients - Exploratory Dataset

The exploratory sample of depressed patients were participants in the Christchurch Outcome of Depression Study (Joyce et al., 2002). This study (conducted by the Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, University of Otago, New Zealand) investigated the long-term outcomes for depressed patients after a randomised trial of two antidepressants (fluoxetine or nortriptyline) (Joyce et al., 2002). Patients were recruited from various sources such as general health practitioners, mental health services, and self-referral. Two criteria were defined for inclusion in the study: that a major depressive episode (defined as Hamilton-17 depression rating scale $\geqslant 14$) was the current principal diagnosis, and the participants were aged between 18 and 64 years old. Patients with serious physical illness, history of schizophrenia, schizoaffective disorder, mania, or from recent antidepressant trials were excluded from the study. Further inclusion criteria were that the patient was drug free (minimum 2 weeks) and not breastfeeding.

Among other information, the Hamilton depression rating scale (27 items), the Symptom Checklist (SCL) and the Temperament and Character Index (TCI) were used to gain information on the personality and symptom severity of the depressed patients. These measurements were repeated after the patients had received treatment for depression. The exploratory sample contains 78 males and 101 females at the baseline measurement. After treatment (six months from the baseline measurement) the sample contains information for 46 males and 68 females.

This data, and subsets of this data, have been used in the following studies. Carter et al. (2000) investigated gender differences in the presentation of a subset of the baseline depressed patients. They found that there were remarkable similarities between males and females in their depressed symptoms. The following significant differences existed. The depressed females reported significantly more carbohydrate craving, appetite increase and weight gain. The depressed females generally were more emotionally expressive of

their depression than the depressed males.

Dysfunctional attitudes and personality in depressed patients were investigated by Luty et al. (1999). Multiple regression analysis found that duration of depression, reward dependence and self directedness explained 45% of the variance of the Dysfunctional Attitudes Scale. Self directedness was the strongest predictor of dysfunctional attitudes.

2.1.2 The Depressed Patients - Confirmatory Dataset

The dataset used for the confirmatory analysis is from the Christchurch Psychotherapy of Depression Study. This study was conducted by the Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences and investigates predictors of response to psychotherapy treatment. This study uses a randomised trial of two psychotherapies (interpersonal psychotherapy and cognitive therapy). The inclusion criteria for the study match that for the Christchurch Outcome of Depression Study.

The confirmatory sample has 46 males and 121 females at baseline. The post treatment measures were conducted 12 months after the baseline measure. There are different sample sizes for the symptom and personality measures at this point. The personality data after treatment has 22 males and 67 females. The symptom data has 33 males and 92 females.

2.1.3 The Normal Volunteers for the Brain Study

This is the first time such a study has been undertaken in Christchurch and possibly in New Zealand. The brain function study required ethical approval from the Canterbury Ethics Committee. As part of the approval process an information sheet was written for potential participants outlining the risks involved with such a study. The information sheet is in Appendix E. Ethical approval was given and the protocol number is 98/05/049.

The decision to use male volunteers only was an important aspect of the study design. With a small sample size of twenty, chosen as the balance between cost and statistical power, focusing on only males removes the potential of gender to confound. Females were excluded as there was the potential for confounding from monthly hormonal fluctuations and the added cost of pregnancy testing required before injection of the radioisotope. Men aged between 20 and 50 years were eligible for the study.

Twenty male volunteers, aged between 20 and 33 years, were recruited from the Christchurch School of Medicine and Health Sciences and the University of Canterbury. All signed informed consent documents. The participants were asked to avoid eating or drinking (other than water) in the hours preceding the scan. They were also asked to avoid the use of drugs, such as painkillers, prior to the scan. Volunteers with a history of alcohol or drug problems, head injury, or mental illness were excluded from the study.

Two volunteers were scanned per evening at the Department of Nuclear Medicine. The volunteers all reported that they were drug free and reported no previous history of alcohol or drug problems, serious head injury or mental illness. On arrival at the scanning facility the volunteers were each put into a quiet room and the cannula needle put in the non-writing arm. A general interview was conducted to obtain background information. Volunteers then completed the symptom checklist and the TCI questionnaire. Ceretec (Amersham Ltd) was used to prepare 99mTc-HMPAO and was donated by Nycomed NZ Ltd. Approximately 500MBq of 99mTc-HMPAO was injected into the volunteer's blood stream whilst they completed the TCI questionnaire (approximately ten minutes into it). Volunteers had been instructed to carry on completing the questionnaire while the radiotracer was injected. SPECT imaging was conducted approximately 30-60 minutes post injection to allow for reduction in the background radiation.

A dual head rotating gamma camera collected data from 64 views (20 seconds per view, step and shoot) around a 180° elliptical arc. A matrix size of 128×128 was chosen with a zoom of two. A high resolution collimator was used and there were weekly calibrations for the centre of rotation. Tomographic reconstruction was completed via filtered back projection (Nowak et al., 1986) (ramp filter, critical frequency 0.5 cycles/cm) with a Butterworth prefilter, and attenuation and uniformity correction. During the reconstruction process the Sorensen attenuation correction method (Petru et al., 1984) was applied within the reconstruction package. The uniformity correction was applied using a cobalt 57 flood source and secondary correction (Amersham, 1994).

2.1.4 The Never Ill Relatives of Bipolar Patients

For comparative purposes a further dataset of the personality profiles of normals has been used. The data is part of the Department of Psychological Medicine's (Christchurch School of Medicine and Health Sciences) South Island Bipolar Study. As part of the study never ill family members of patients were asked to complete the TCI questionnaire creating a dataset of normals. This dataset contains personality information on 36 females and 37 males.

2.2 Descriptive Statistics

This section presents some descriptive statistics for the datasets and makes some initial comparisons across gender, across time points and across the datasets. Each trait and symptom is presented and the distributions across the groups discussed. Side-by-side box plots are used to compare the levels of traits and symptoms within each group. Following on from this, hypothesis tests are conducted to investigate differences in the median SCL

and TCI levels of the groups on each variable and across time in the depressed dataset. For brevity T0, T6 and T12 will be used to describe the baseline, six month and twelve month time points respectively.

2.2.1 Personality

This section investigates the distribution of the personality scores for the depressed patients and normals. The depressed patients have data both pre and post treatment allowing for investigation of changes in the personality traits that may relate to the expected improvement in the depression symptoms with treatment. When comparing the depressed patients to the normals it is advisable to remember that with these small sample sizes, comparisons are only valid for these samples and are not representative of the general population.

Table 2.1 presents the value for the median, lower quartile and upper quartile for each group for the temperament traits and Table 2.2 presents the median, lower quartile and upper quartile values for each group for the character traits. Harm avoidance medians are lower in the normal groups than the depressed patients and self directedness scores are higher in the never ill relatives of bipolar patients than the depressed patients. This is similar to the pattern seen with the upper quartile values. The normal groups have lower upper quartile values than the depressed patients for harm avoidance and the normals from the bipolar study have higher self directedness than the depressed patients. For novelty seeking, the normal brain study males have a higher lower quartile value than the depressed patients and the never ill males and females have lower novelty seeking values than the depressed patients. The depressed patients harm avoidance scores, at both time points, are higher than the three normal groups. Persistence lower quartile values are lower in the depressed patients compared to the normal males from the brain study and the bipolar study. Self directedness lower quartile values are higher in the normal groups with the bipolar normal groups higher than the males from the brain study.

Table 2.3 presents the mean, median and skewness values for the personality variables. Harm Avoidance, cooperativeness and self directedness are generally skewed across most of the groups. The never-ill males have a large degree of skew on the character traits.

The frequency distributions for novelty seeking are presented in this section (Figure 2.1) as an example. These distributions show that in general the data is not normal and is often highly skewed.

Novelty seeking scores for the depressed patients appear to be symmetrically distributed. The distribution appears similar pre and post treatment. The brain study normal males appear to have values of novelty seeking quite close to the median, with no values below 0.4. This is not unexpected, as people low in novelty seeking would not tend

Group		Temperament			
		NS	HA	RD	P
Exploratory	Dep Fem T0	0.53 (0.41, 0.66)	0.74 (0.59, 0.83)	0.66 (0.53, 0.78)	0.50 (0.25, 0.75)
	Dep Male T0	0.50 (0.41, 0.60)	0.66 (0.51, 0.85)	0.58 (0.49, 0.67)	0.50 (0.38, 0.75)
	Dep Fem T6	0.53 (0.41, 0.62)	0.63 (0.48, 0.74)	0.72 (0.61, 0.83)	0.50 (0.38, 0.75)
	Dep Male T6	0.52 (0.41, 0.63)	0.61 (0.39, 0.80)	0.59 (0.50, 0.71)	0.63 (0.38, 0.75)
Confirmatory	Dep Fem T0	0.49 (0.39, 0.58)	0.73 (0.60, 0.78)	0.71 (0.60, 0.78)	0.44 (0.31, 0.59)
	Dep Male T0	0.46 (0.37, 0.60)	0.74 (0.56, 0.83)	0.51 (0.38, 0.71)	0.39 (0.27, 0.52)
	Dep Fem T12	0.50 (0.39, 0.61)	0.65 (0.49, 0.74)	0.74 (0.64, 0.84)	0.47 (0.29, 0.67)
	Dep Male T12	0.53 (0.42, 0.59)	0.67 (0.53, 0.69)	0.55 (0.38, 0.78)	0.50 (0.39, 0.64)
Normal	Brain Males	0.53 (0.48, 0.61)	0.40 (0.31, 0.46)	0.55 (0.45, 0.60)	0.56 (0.51, 0.66)
	NI Males	0.42 (0.35, 0.53)	0.35 (0.22, 0.44)	0.63 (0.49, 0.77)	0.65 (0.55, 0.76)
	NI Females	0.43 (0.30, 0.53)	0.40 (0.27, 0.53)	0.72 (0.67, 0.79)	0.49 (0.39, 0.62)

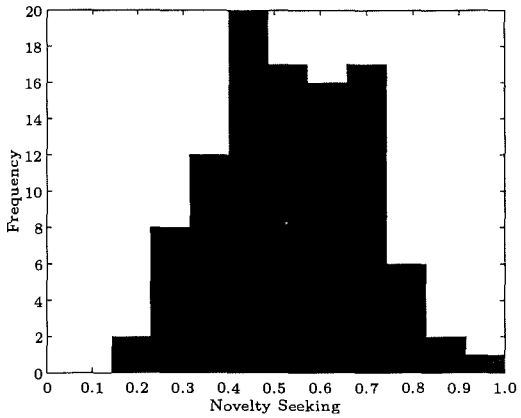
Table 2.1: Median (lower quartile, upper quartile) values for the personality scores for the temperament traits.

Group		Character		
		S	C	ST
Exploratory	Dep Fem T0	0.53 (0.39, 0.71)	0.83 (0.72, 0.88)	0.28 (0.19, 0.44)
	Dep Male T0	0.50 (0.37, 0.64)	0.73 (0.64, 0.82)	0.30 (0.19, 0.48)
	Dep Fem T6	0.71 (0.51, 0.84)	0.87 (0.77, 0.91)	0.28 (0.18, 0.43)
	Dep Male T6	0.59 (0.44, 0.76)	0.78 (0.69, 0.88)	0.32 (0.21, 0.54)
Confirmatory	Dep Fem T0	0.56 (0.39, 0.68)	0.83 (0.74, 0.89)	0.25 (0.16, 0.42)
	Dep Male T0	0.47 (0.32, 0.64)	0.75 (0.63, 0.83)	0.23 (0.16, 0.39)
	Dep Fem T12	0.70 (0.49, 0.82)	0.87 (0.82, 0.93)	0.30 (0.17, 0.49)
	Dep Male T12	0.55 (0.45, 0.79)	0.78 (0.64, 0.87)	0.31 (0.16, 0.41)
Normal	Brain Males	0.61 (0.56, 0.71)	0.66 (0.59, 0.75)	0.31 (0.26, 0.47)
	NI Males	0.89 (0.84, 0.95)	0.88 (0.80, 0.95)	0.22 (0.16, 0.32)
	NI Females	0.85 (0.79, 0.93)	0.90 (0.83, 0.94)	0.24 (0.18, 0.43)

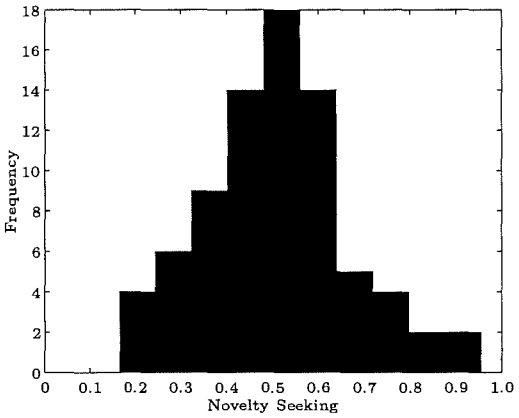
Table 2.2: Median (lower quartile, upper quartile) values for the personality scores for the character traits.

Group		Statistic	Temperament				Character		
			NS	HA	RD	P	S	C	ST
Exploratory	Dep Fem T0	Mean	0.53	0.70	0.65	0.53	0.54	0.79	0.31
		Median	0.53	0.74	0.66	0.50	0.53	0.83	0.28
		Skewness	0.05	**-1.09**	-0.39	0.28	-0.15	**-0.98**	**0.56**
	Dep Fem T6	Mean	0.51	0.60	0.70	0.52	0.66	0.83	0.31
		Median	0.53	0.63	0.72	0.50	0.71	0.87	0.28
		Skewness	-0.12	-0.49	**-0.72**	0.17	-0.42	**-1.19**	**0.50**
	Dep Male T0	Mean	0.51	0.65	0.58	0.55	0.51	0.72	0.34
		Median	0.50	0.66	0.58	0.50	0.50	0.73	0.30
		Skewness	0.31	**-0.56**	-0.28	0.12	0.04	**-0.75**	**0.56**
	Dep Male T6	Mean	0.50	0.59	0.60	0.60	0.60	0.77	0.38
		Median	0.52	0.61	0.59	0.63	0.59	0.78	0.32
		Skewness	-0.14	-0.34	-0.07	0.01	-0.14	**-0.97**	**0.58**
Confirmatory	Dep Fem T0	Mean	0.49	0.71	0.69	0.45	0.54	0.80	0.30
		Median	0.49	0.73	0.71	0.44	0.56	0.83	0.25
		Skewness	0.13	**-0.91**	-0.45	0.20	-0.08	**-0.93**	**0.88**
	Dep Fem T12	Mean	0.50	0.62	0.72	0.48	0.65	0.85	0.34
		Median	0.50	0.65	0.74	0.47	0.70	0.87	0.30
		Skewness	0.03	-0.31	**-0.88**	0.12	-0.22	**-1.55**	**0.62**
	Dep Male T0	Mean	0.47	0.69	0.52	0.42	0.48	0.71	0.30
		Median	0.46	0.74	0.51	0.39	0.47	0.75	0.23
		Skewness	0.18	**-0.68**	-0.05	**0.52**	0.24	**-1.03**	**0.91**
	Dep Male T12	Mean	0.49	0.63	0.57	0.50	0.59	0.76	0.28
		Median	0.53	0.67	0.55	0.50	0.55	0.78	0.31
		Skewness	**-0.79**	**-0.55**	-0.10	-0.27	-0.17	-0.33	-0.18
Normal	NI Females	Mean	0.42	0.42	0.72	0.51	0.84	0.87	0.33
		Median	0.43	0.40	0.72	0.49	0.85	0.90	0.24
		Skewness	-0.45	0.21	**-1.19**	-0.05	**-0.59**	**-1.99**	**0.78**
	NI Males	Mean	0.45	0.32	0.63	0.67	0.86	0.85	0.27
		Median	0.42	0.35	0.63	0.65	0.89	0.88	0.22
		Skewness	**0.74**	-0.45	**-0.51**	-0.03	**-2.62**	**-1.42**	**1.07**
	Brain Males	Mean	0.55	0.39	0.52	0.57	0.64	0.67	0.37
		Median	0.53	0.40	0.55	0.56	0.61	0.66	0.31
		Skewness	**0.68**	**-0.65**	**-0.61**	-0.38	0.48	-0.13	**0.79**

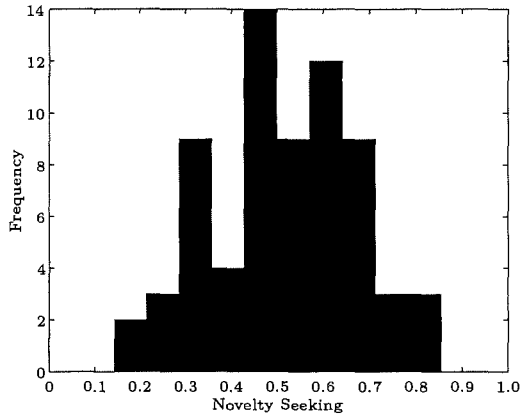
Table 2.3: Mean, median and skewness values for the personality traits.



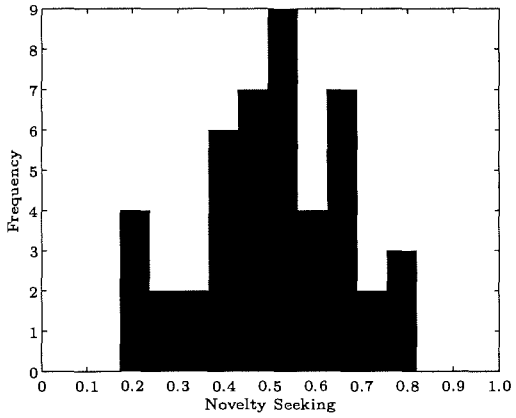
(a) Depressed Females at Baseline



(b) Depressed Males at Baseline

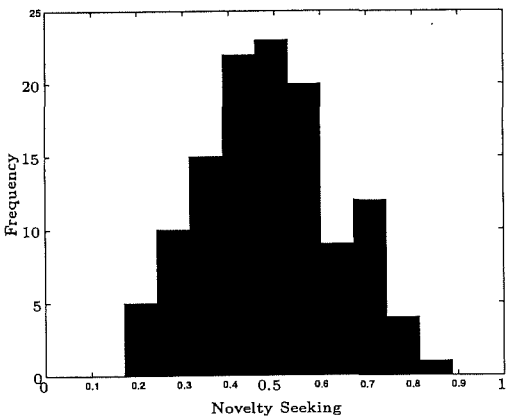


(c) Depressed Females at Six Months

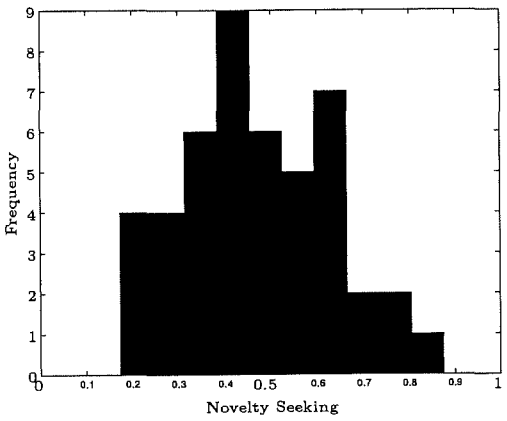


(d) Depressed Males at Six Months

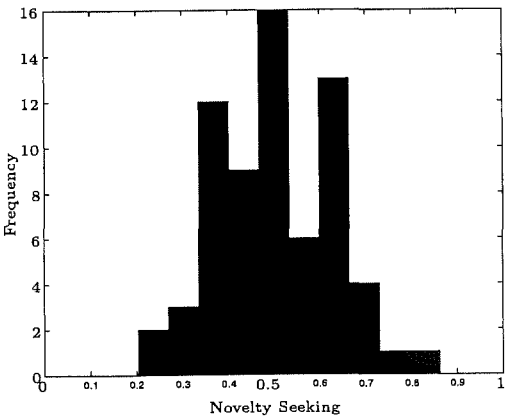
Figure 2.1: Frequency distributions for novelty seeking for the depressed patients (exploratory sample).



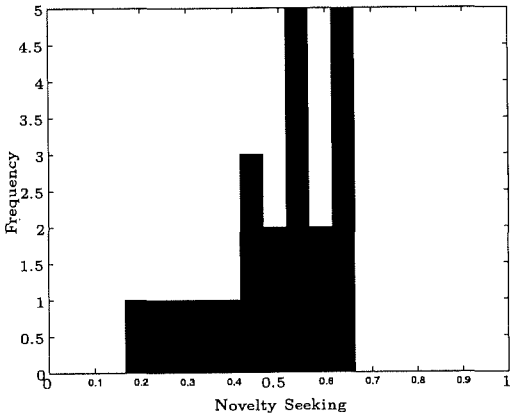
(a) Depressed Females at Baseline



(b) Depressed Males at Baseline

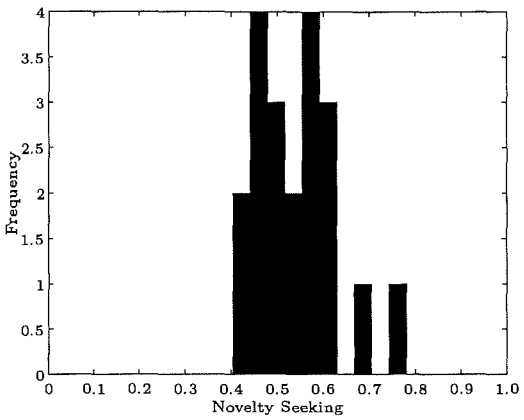


(c) Depressed Females at Twelve Months

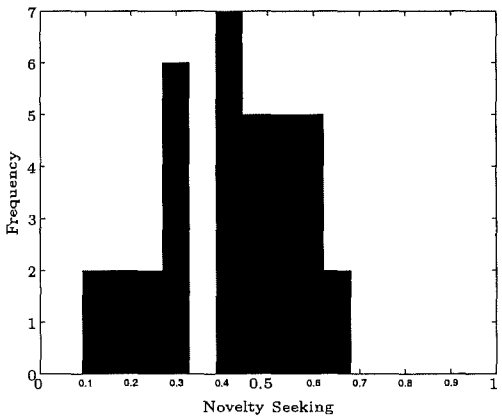


(d) Depressed Males at Twelve Months

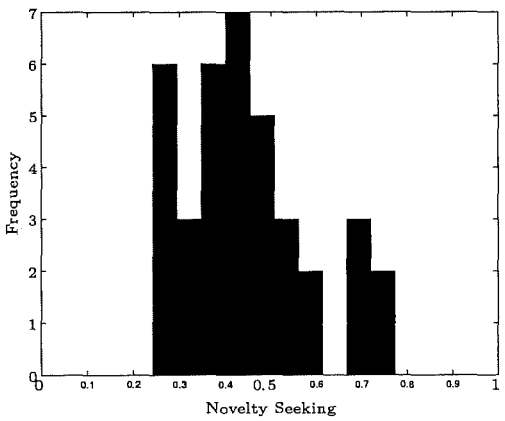
Figure 2.2: Frequency distributions for novelty seeking for the depressed patients (confirmatory sample).



(a) Brain Study Normal Males



(b) Never Ill Female Relatives of Bipolar Patients



(c) Never Ill Male Relatives of Bipolar Patients

Figure 2.3: Frequency distributions for novelty seeking for the normal groups.

to volunteer for the brain study.

The baseline measure of harm avoidance in the depressed females has a distribution of relatively high scores. After treatment the harm avoidance scores for the females have a smaller maximum. The depressed males also have high scores on harm avoidance. In comparison the normal brain study males are low in harm avoidance with all scores below 0.53. The nature of the brain study may bias the range of harm avoidance values as a person high in harm avoidance is unlikely to volunteer for this study.

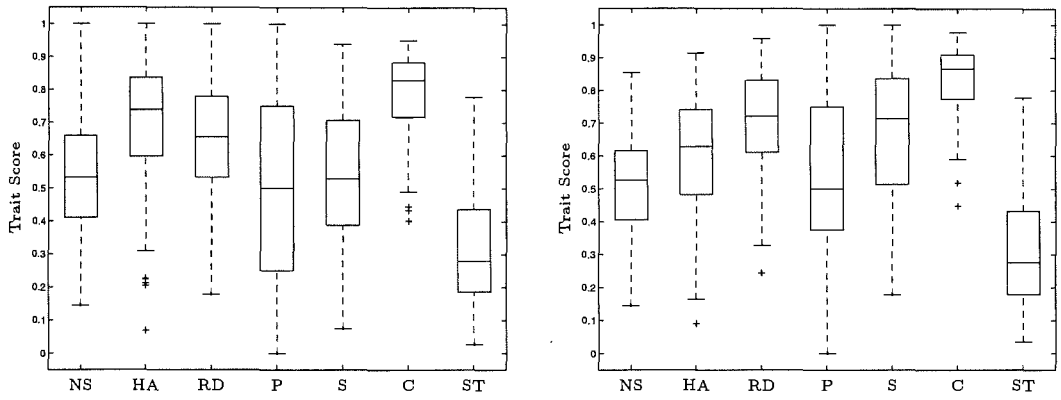
The depressed females have high reward dependence scores, with 79% of the scores above 0.5 at baseline for the exploratory data. This increased to 88% after treatment. The males, whilst high scorers, exhibit a much smaller increase in reward dependence values. At baseline 73% of the scores are greater than 0.5, for the exploratory data, and six months later this has increased to 76%. The normal brain study males are localised around the central reward dependence scores with no extreme values, 65% of the reward dependence scores are greater than 0.5.

The exploratory sample of depressed females persistence scores at baseline have a bimodal distribution. The distribution appears similar at six months. The depressed males (exploratory sample) have a trimodal distribution with peaks at 0.3, 0.55 and 0.85. The distribution appears similar at six months. The normal brain study males persistence values appear to cluster around a value of about 0.55 with less extreme values compared to the depressives.

Self directedness values for the depressed females (exploratory sample) at baseline appear to be symmetrically distributed around a value of 0.5. After treatment there is a shift towards increased values. The depressed males (exploratory sample) exhibit a similar distribution at baseline with the same upwards shift six months later. The normal brain study males have a higher distribution of self directedness values than the depressives with a minimum value of 0.5. At baseline both the exploratory samples of depressed females and males appear highly cooperative. The distribution is left skewed and remains similar six months later. The normal males also appear highly cooperative with a minimum value of 0.5, however the shape is not skewed. The distributions of self transcendence scores for the exploratory depressed males and females are right skewed and do not exhibit any obvious changes across time. The depressives are low in self transcendence with most values less than 0.5. The normal brain study males are also low in self transcendence with a right skewed distribution.

Summary of Personality Score Distributions

A comparison of the score distributions for the depressed females are presented in Figures 2.4 and 2.6 showing both the before and after treatment distributions for the exploratory and confirmatory groups. These side-by-side box plots show that the depressed females



(a) Baseline (b) Six Months

Figure 2.4: Distribution of personality scores for the depressed females (exploratory sample).

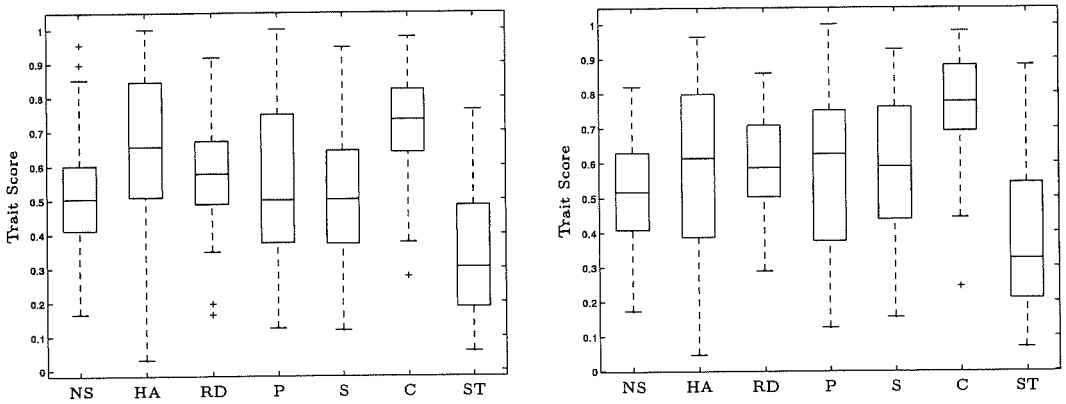
are highly cooperative and harm avoiding, and low in self transcendence at baseline. After treatment harm avoidance decreases, and reward dependence and self directedness increase. The distribution of persistence shows a substantial increase in the lower quartile value but there are no corresponding increases to the maximum, upper quartile, median or minimum value. The females are highly cooperative and reward dependent, with low self transcendence levels after treatment.

Figures 2.5 and 2.7 present side-by-side box plots of the personality scores for the exploratory and confirmatory groups of depressed males at baseline and after treatment. The males at baseline are highly cooperative and harm avoiding, and low in self transcendence. After treatment self directedness has increased. The median value for persistence increases substantially but the maximum value, upper quartile, lower quartile and minimum stay at similar levels. After treatment, the males are highly cooperative and low in self transcendence. Some of the traits have potential outlier measures.

Figure 2.8 presents side-by-side box plots for the personality traits for the normal groups. The normal males from the brain study are highly cooperative and low in self transcendence. The never ill male and female relatives of bipolar patients are particularly high in self directedness and cooperativeness. They are also low in harm avoidance.

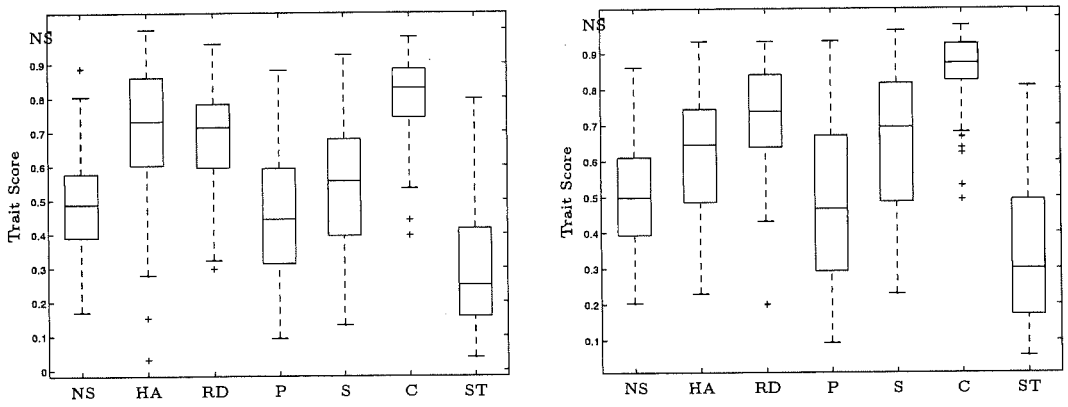
Personality Characteristics for the Median Person in Each Group

The depressed female is typically high in harm avoidance, reward dependence, cooperativeness and low in self transcendence. After treatment they are high in reward dependence, self directedness and cooperativeness and low in self transcendence. The normal



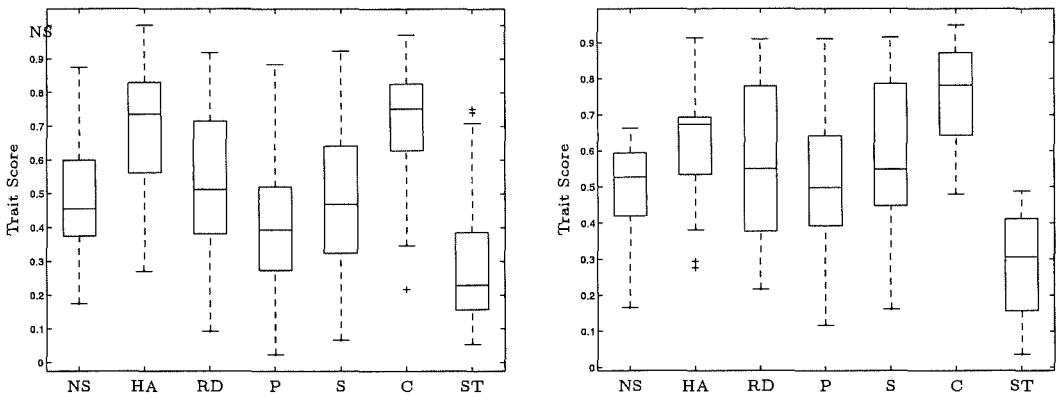
(a) Baseline (b) Six Months

Figure 2.5: Distribution of personality scores for the depressed males (exploratory sample).



(a) Baseline (b) Twelve Months

Figure 2.6: Distribution of personality scores for the depressed females (confirmatory sample).



(a) Baseline

(b) Twelve Months

Figure 2.7: Distribution of personality scores for the depressed males (confirmatory sample).

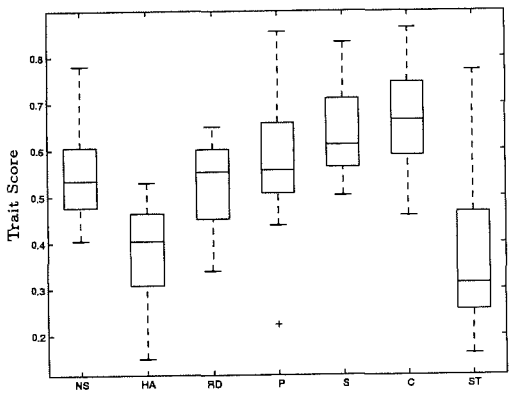
females from the bipolar study are high in reward dependence, self directedness and cooperativeness and low in self transcendence.

The depressed male, at baseline, typically has high harm avoidance and cooperativeness combined with low self transcendence. After treatment the typical depressed male has high cooperativeness and low self transcendence. The normal males from the brain study and the bipolar study both have high levels of self directedness and cooperativeness, and low levels of harm avoidance and self transcendence.

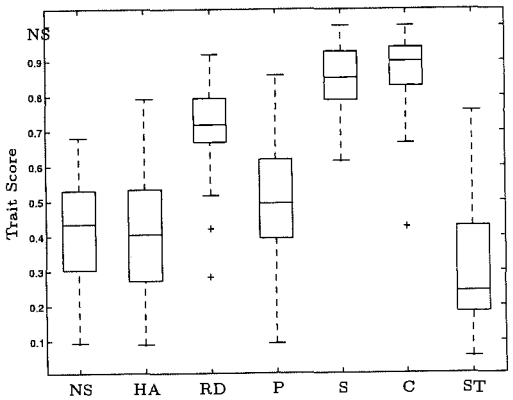
2.2.2 Symptoms of Depression

The next section investigates the distribution of the symptom scores, as measured by the Symptom Checklist, for the depressed males, depressed females and normal males. It is expected that the symptoms in the depressed patients will reduce substantially after treatment for depression. The normal brain study males are expected to have very low symptom scores, as they are not suffering from any psychiatric illness that would lead to psychological distress.

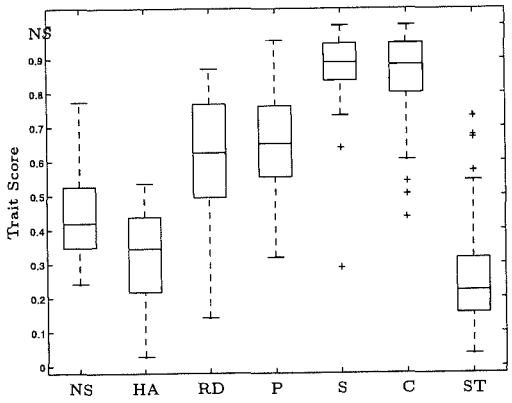
The median symptom values for each group are presented in Table 2.4. The depression symptom is the highest for the exploratory and confirmatory datasets of depressed patients at baseline. After treatment obsessive compulsive symptoms are the highest for the exploratory dataset and depression has the highest median for the confirmatory dataset. The normal males have obsessive compulsive symptoms as the highest median value. For the upper quartile values depression is the highest value for the exploratory and confirmatory datasets at both time points. The normal males from the brain study also



(a) Brain Study Normal Males



(b) Never Ill Female Relatives of Bipolar Patients



(c) Never Ill Male Relatives of Bipolar Patients

Figure 2.8: Distribution of personality scores for the normal groups.

have depression as the highest upper quartile value. At baseline the depressed patients highest symptom, on the lower quartile, was depression followed by obsessive compulsive symptoms. After treatment, anxiety was the highest lower quartile value for the exploratory sample of depressed patients and for the confirmatory sample of depressed patients depression was still the highest symptom. For the normal males from the brain study, obsessive compulsive symptoms were the highest lower quartile value.

The frequency distributions were plotted for the somatisation symptom scores for each of the five groups, depressed males and females (exploratory sample), depressed males and females (confirmatory sample), and the normal males from the brain study (Figures 2.9, 2.10 and 2.11). The depressed males and females exhibit a similar right skewed distribution at baseline. After treatment, the somatisation symptoms are reduced and the distribution remains right skewed. The normal males have much lower somatisation scores than the depressed patients both pre and post treatment. The distributions are often left truncated as the scores start at zero. The frequency distributions also show that, in general, the depressed males post treatment symptoms are usually higher than the depressed females post treatment symptoms.

The mean, median and skewness are presented in Table 2.5 and show that in there is more skew in the data after treatment for the depressed patients. The normal males from the brain study have skewed distributions for the symptoms.

Figure 2.12 presents side-by-side box plots for the exploratory sample of depressed females symptoms, pre and post treatment. At baseline (Figure 2.12(a)) the highest symptom is depression and the lowest symptom is phobic anxiety. After treatment (Figure 2.12) all the symptoms scores have reduced, obsessive compulsive has the highest median, but depression has the highest upper quartile and maximum. Phobic anxiety still appears to be the lowest symptom.

Side-by-side box plots for the exploratory group of depressed males symptoms are presented in Figure 2.13. At baseline (Figure 2.13(a)) depression is the highest scoring symptom and phobic anxiety, like the females, is the lowest. The symptoms are reduced after treatment (Figure 2.13(b)), with depression the highest symptom and phobic anxiety the lowest symptom.

A similar pattern is seen for the confirmatory dataset presented in Figures 2.14 and 2.15. The pattern of symptoms in the exploratory dataset appears similar for the males and females at baseline. After treatment the female's symptoms have reduced more than the male's symptoms. For the confirmatory dataset, the baseline symptom pattern appears similar across males and females. Again the female's symptoms improve more after treatment, than the males.

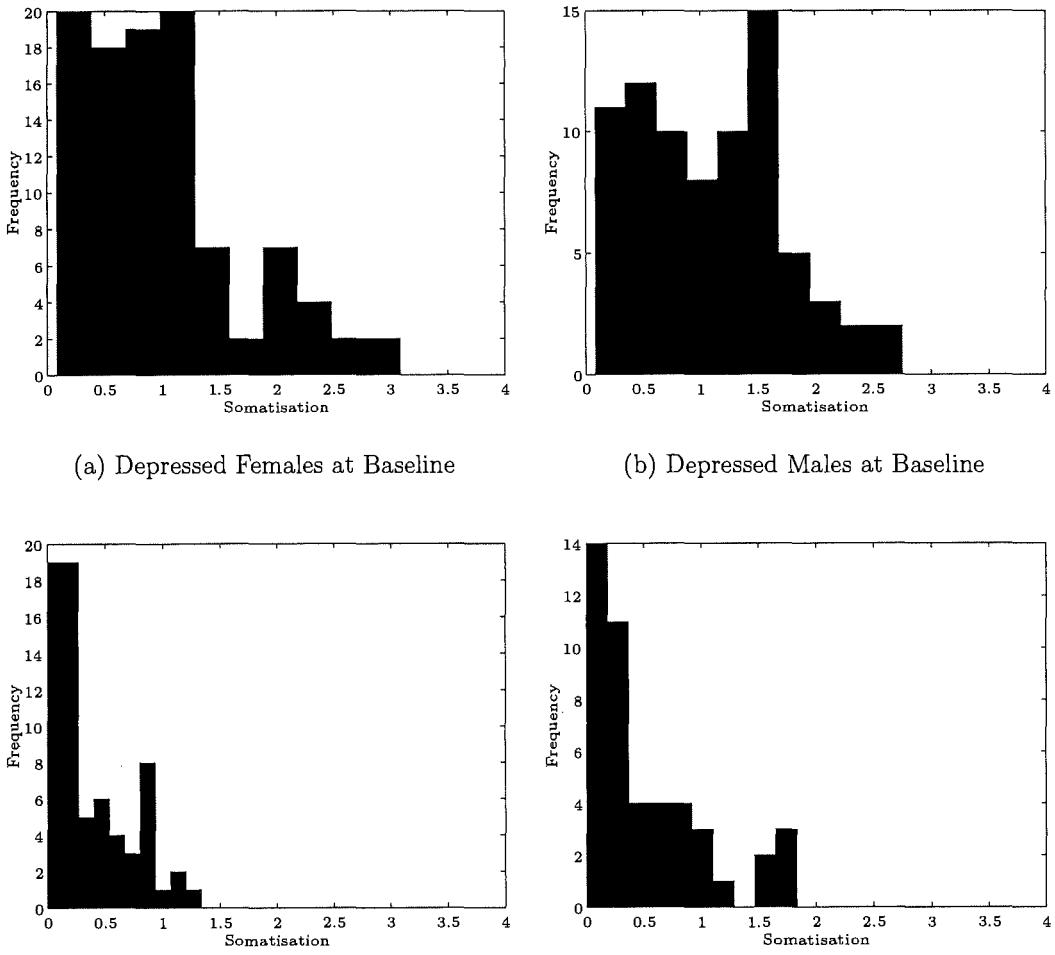
The normal male's symptoms are presented in Figure 2.16. Obsessive compulsive has the highest median and depression the highest upper quartile. Phobic anxiety is the lowest

Group		S	OC	IS
Exploratory	Dep Fem T0	0.83 (0.50, 1.25)	1.90 (1.28, 2.33)	1.56 (1.00, 2.33)
	Dep Male T0	1.00 (0.50, 1.50)	2.15 (1.40, 2.50)	1.67 (1.22, 2.33)
	Dep Fem T6	0.25 (0.08, 0.67)	0.50 (0.10, 0.90)	0.22 (0.00, 0.83)
	Dep Male T6	0.33 (0.08, 0.83)	0.90 (0.20, 1.50)	0.61 (0.11, 1.44)
Confirmatory	Dep Fem T0	0.75 (0.42, 1.33)	1.50 (1.00, 2.00)	1.44 (0.89, 2.33)
	Dep Male T0	0.67 (0.33, 1.25)	1.80 (1.10, 2.30)	1.11 (0.78, 1.78)
	Dep Fem T12	0.17 (0.08, 0.63)	0.30 (0.10, 0.75)	0.22 (0.11, 0.67)
	Dep Male T12	0.25 (0.00, 0.77)	0.60 (0.28, 1.10)	0.56 (0.11, 0.83)
Normal	Brain Males	0.17 (0.08, 0.25)	0.30 (0.15, 0.40)	0.11 (0.06, 0.39)
Group		D	A	AH
Exploratory	Dep Fem T0	2.50 (1.92, 2.94)	1.50 (0.90, 2.00)	1.00 (0.67, 1.67)
	Dep Male T0	2.46 (1.85, 2.85)	1.60 (1.00, 2.10)	1.17 (0.83, 1.83)
	Dep Fem T6	0.46 (0.08, 1.15)	0.30 (0.10, 0.60)	0.17 (0.00, 0.42)
	Dep Male T6	0.92 (0.23, 1.67)	0.53 (0.30, 1.10)	0.17 (0.00, 0.83)
Confirmatory	Dep Fem T0	2.15 (1.46, 2.63)	1.20 (0.78, 1.73)	0.83 (0.46, 1.50)
	Dep Male T0	1.88 (1.38, 2.31)	1.05 (0.60, 1.60)	0.67 (0.33, 1.33)
	Dep Fem T12	0.38 (0.15, 0.96)	0.30 (0.10, 0.50)	0.17 (0.00, 0.33)
	Dep Male T12	0.77 (0.29, 1.38)	0.50 (0.20, 0.63)	0.17 (0.17, 0.50)
Normal	Brain Males	0.15 (0.08, 0.42)	0.10 (0.00, 0.40)	0.17 (0.00, 0.33)
Group		PA	PI	P
Exploratory	Dep Fem T0	0.43 (0.14, 1.14)	1.00 (0.50, 1.67)	0.90 (0.50, 1.40)
	Dep Male T0	0.71 (0.14, 1.29)	1.25 (0.67, 2.17)	1.10 (0.70, 1.63)
	Dep Fem T6	0.00 (0.00, 0.14)	0.17 (0.00, 0.42)	0.10 (0.00, 0.40)
	Dep Male T6	0.00 (0.00, 0.29)	0.50 (0.00, 1.00)	0.30 (0.00, 0.50)
Confirmatory	Dep Fem T0	0.43 (0.00, 0.71)	1.00 (0.50, 1.50)	0.70 (0.40, 1.13)
	Dep Male T0	0.29 (0.00, 0.71)	1.00 (0.33, 1.50)	0.80 (0.50, 1.30)
	Dep Fem T12	0.00 (0.00, 0.14)	0.00 (0.00, 0.33)	0.05 (0.00, 0.30)
	Dep Male T12	0.00 (0.00, 0.14)	0.17 (0.00, 0.67)	0.30 (0.00, 0.50)
Normal	Brain Males	0.00 (0.00, 0.07)	0.00 (0.00, 0.33)	0.00 (0.00, 0.15)

Table 2.4: Median (lower quartile, upper quartile) values for the symptom scores.

Group		Statistic	S	OC	IS	D	A	AH	PA	PI	P
Exploratory	Dep Fem T0	Mean	1.01	1.83	1.70	2.43	1.50	1.23	0.73	1.11	0.99
		Median	0.83	1.90	1.56	2.50	1.50	1.00	0.43	1.00	0.90
		Skewness	**1.00**	-0.11	0.27	-0.46	0.40	**0.97**	**1.37**	0.35	0.37
	Dep Fem T6	Mean	0.38	0.58	0.46	0.69	0.40	0.33	0.14	0.30	0.21
		Median	0.25	0.50	0.22	0.46	0.30	0.17	0.00	0.17	0.10
		Skewness	**0.87**	**1.58**	**0.82**	**1.08**	**1.56**	**2.60**	**2.45**	**2.04**	**1.02**
	Dep Male T0	Mean	1.07	2.03	1.75	2.33	1.56	1.38	0.79	1.39	1.17
		Median	1.00	2.15	1.67	2.46	1.60	1.17	0.71	1.25	1.10
		Skewness	0.39	-0.04	0.23	**-0.63**	0.10	**0.67**	**1.01**	0.21	0.35
	Dep Male T6	Mean	0.53	0.89	0.85	0.99	0.73	0.50	0.30	0.56	0.40
		Median	0.33	0.90	0.61	0.92	0.53	0.17	0.00	0.50	0.30
		Skewness	**1.13**	0.43	**0.98**	0.47	**1.22**	**1.76**	**2.20**	**0.98**	**1.32**
Confirmatory	Dep Fem T0	Mean	0.94	1.55	1.62	2.07	1.29	1.05	0.58	1.09	0.82
		Median	0.75	1.50	1.44	2.15	1.20	0.83	0.43	1.00	0.70
		Skewness	**0.99**	**0.67**	**0.58**	-0.20	**0.53**	**1.04**	**2.00**	**0.73**	**1.20**
	Dep Fem T12	Mean	0.38	0.50	0.50	0.66	0.40	0.30	0.16	0.30	0.20
		Median	0.17	0.30	0.22	0.38	0.30	0.17	0.00	0.00	0.05
		Skewness	**1.77**	**1.38**	**1.74**	**1.58**	**1.50**	**2.88**	**3.33**	**2.48**	**2.64**
	Dep Male T0	Mean	0.86	1.71	1.31	1.88	1.16	0.89	0.48	1.01	0.92
		Median	0.67	1.80	1.11	1.88	1.05	0.67	0.29	1.00	0.80
		Skewness	**1.22**	0.39	**0.58**	0.09	**0.83**	**0.92**	**1.45**	**0.62**	**1.14**
	Dep Male T12	Mean	0.45	0.76	0.63	0.84	0.48	0.34	0.15	0.39	0.37
		Median	0.25	0.60	0.56	0.77	0.50	0.17	0.00	0.17	0.30
		Skewness	**1.16**	**1.14**	**0.83**	**0.54**	**0.95**	**1.51**	**3.16**	**0.74**	**1.28**
Normal	Brain Males	Mean	0.20	0.31	0.27	0.26	0.23	0.22	0.05	0.21	0.11
		Median	0.17	0.30	0.11	0.15	0.10	0.17	0.00	0.00	0.00
		Skewness	**0.76**	0.35	**1.33**	**0.84**	**0.96**	**0.66**	**1.64**	**1.65**	**2.36**

Table 2.5: Mean, median and skewness values for the symptom scores.



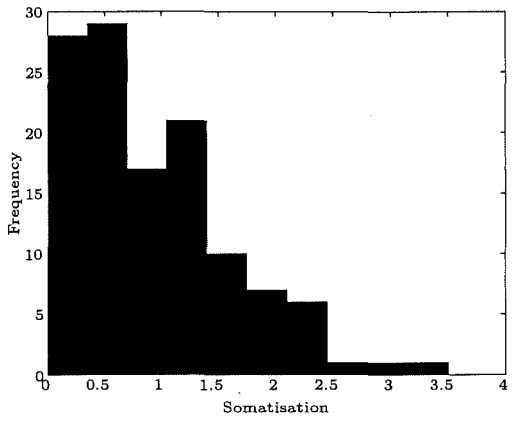
(a) Depressed Females at Baseline

(b) Depressed Males at Baseline

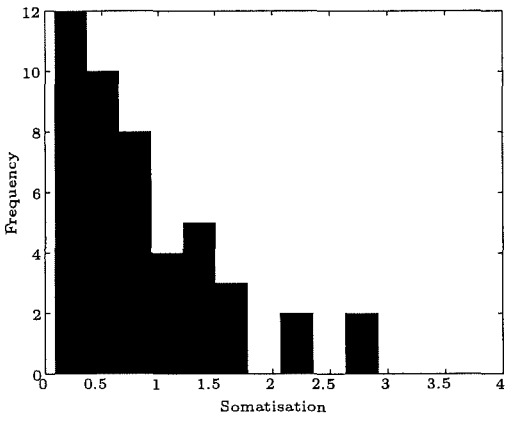
(c) Depressed Females at Six Months

(d) Depressed Males at Six Months

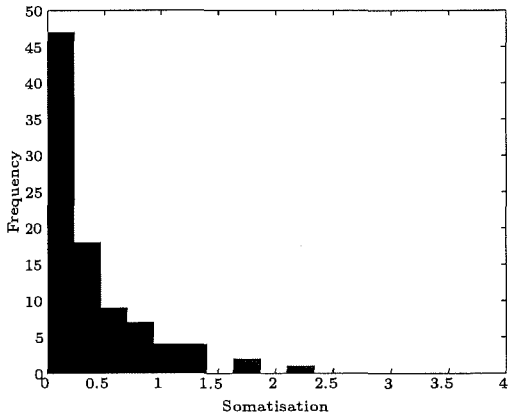
Figure 2.9: Frequency distributions of somatisation scores for the exploratory dataset.



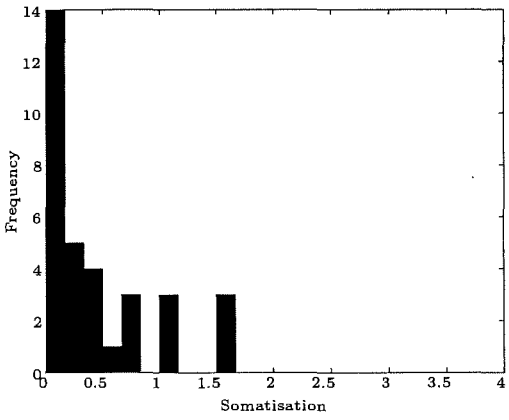
(a) Depressed Females at Baseline



(b) Depressed Males at Baseline

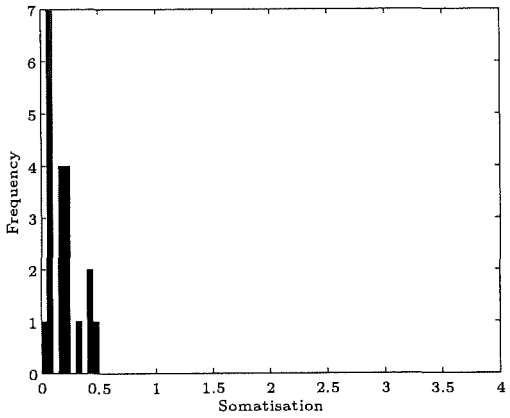


(c) Depressed Females at Twelve Months



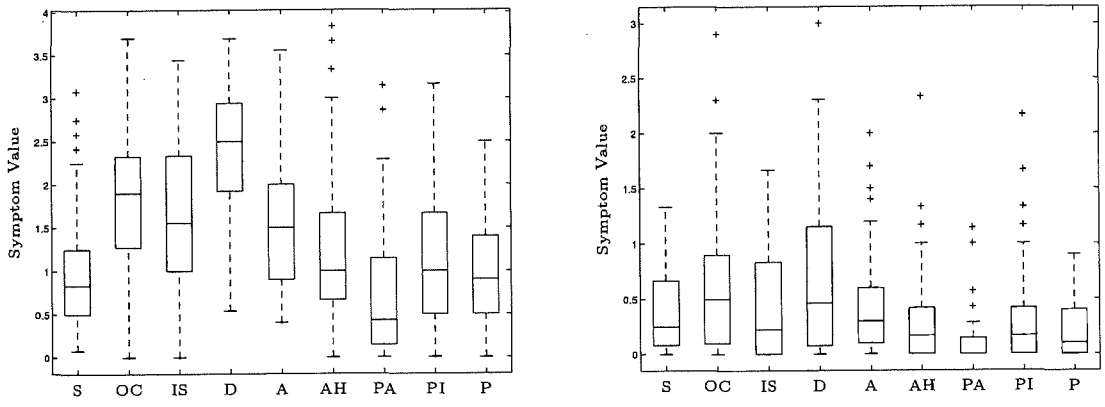
(d) Depressed Males at Twelve Months

Figure 2.10: Frequency distributions of somatisation scores for the confirmatory dataset.



(a) Brain Study Normal Males

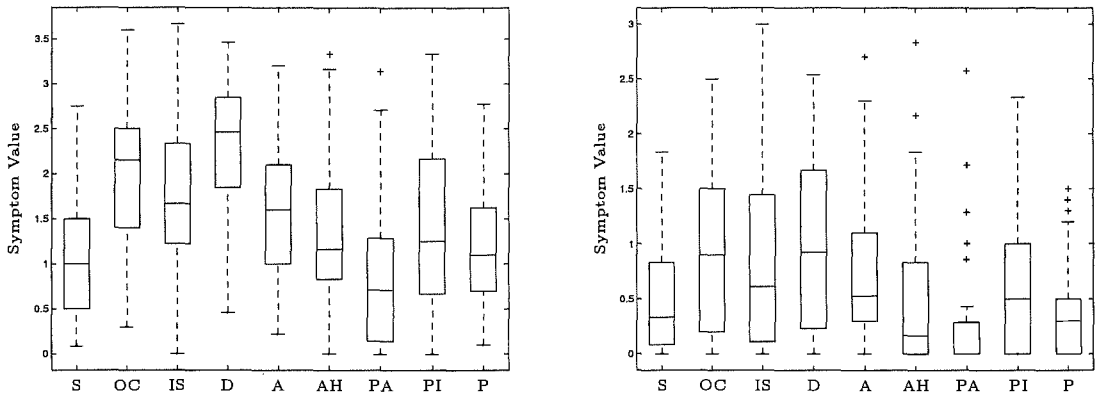
Figure 2.11: Frequency Distributions of Somatisation Scores (for comparison with Figures 2.9 and 2.10)



(a) Baseline

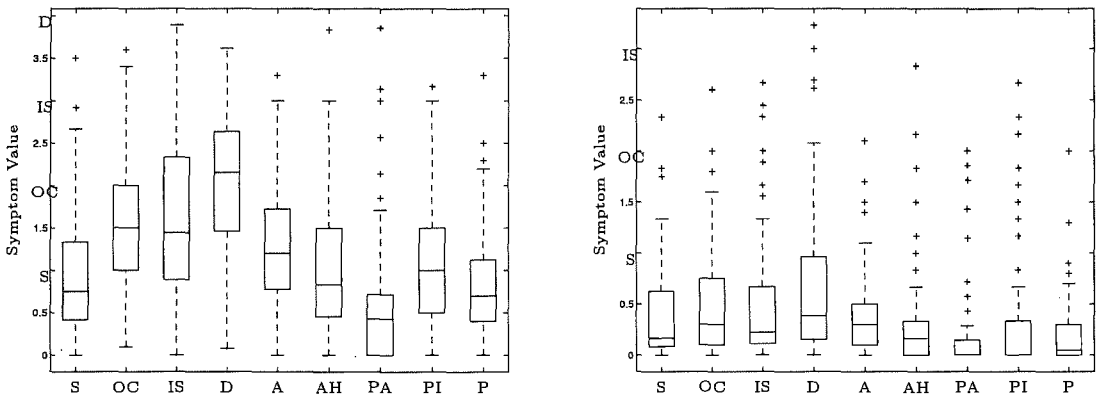
(b) Six Months

Figure 2.12: Boxplots showing distribution of symptom scores for the depressed females (exploratory sample).



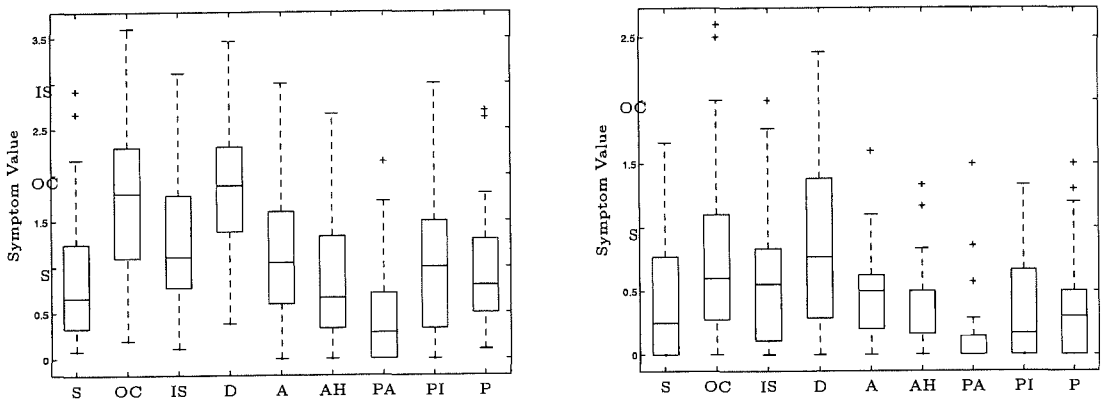
(a) Baseline (b) Six Months

Figure 2.13: Boxplots showing distribution of symptom scores for the depressed males (exploratory sample).



(a) Baseline (b) Twelve Months

Figure 2.14: Boxplots showing distribution of symptom scores for the depressed females (confirmatory sample).



(a) Baseline

(b) Twelve Months

Figure 2.15: Boxplots showing distribution of symptom scores for the depressed males (confirmatory sample).

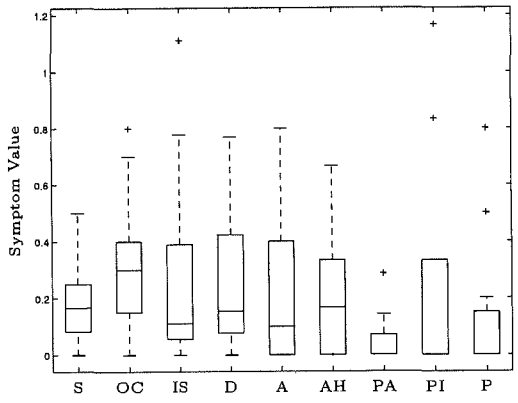
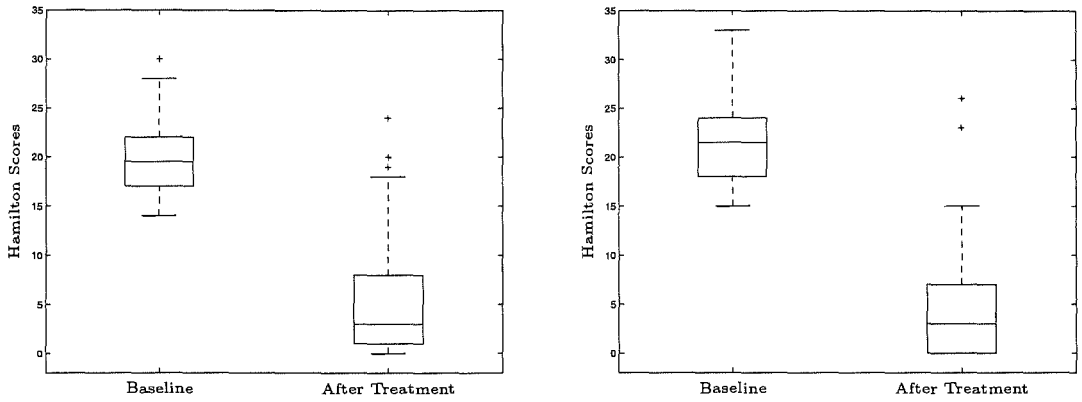


Figure 2.16: Boxplots showing distribution of symptom scores for the normal males.

scoring symptom. The symptoms of the depressed patients, after treatment, are higher than those of the normal males from the brain study.

2.3 Hamilton Depression Rating

The Hamilton Depression Ratings were available for the exploratory data. Figure 2.17 presents boxplots of the distribution of the Hamilton scores for the exploratory males and females before and after treatment. The box plots show that the males appear to have more severe depression at baseline and are more improved compared to the females, after treatment. This is again seen in the boxplots of the change in Hamilton score (Figure 2.18). The males have a more negative change in Hamilton score than the females. The



(a) Female Hamilton Scores

(b) Male Hamilton Scores

Figure 2.17: Boxplots showing the distribution of the Hamilton scores for the exploratory dataset.

change in Hamilton score is calculated as the after treatment score minus the baseline score, this means that a more negative score is a larger improvement. Thus the males appear to have improved more than the females.

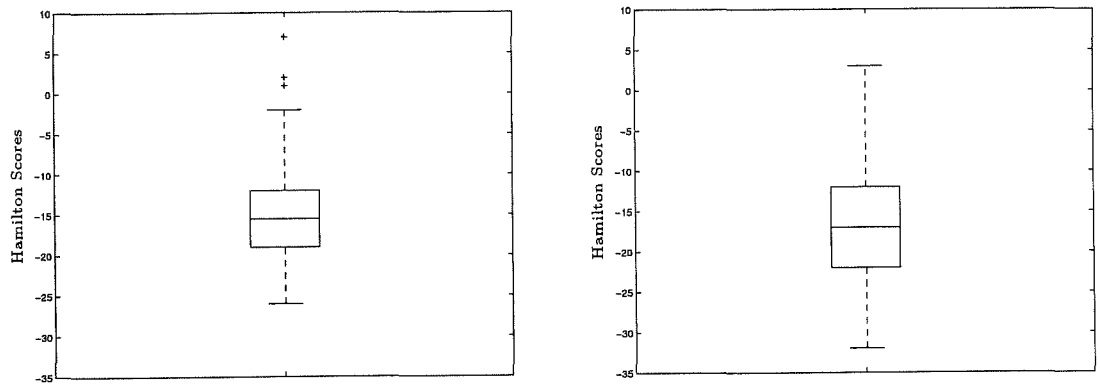
2.4 Hypothesis Testing

The following section investigates the similarities and differences between, firstly, the two depressed groups (exploratory and confirmatory) and secondly, the three normal groups (brain volunteers, and the bipolar study normal males and females). Differences across time and gender are investigated. Due to the skewness demonstrated in some of the data, nonparametric hypothesis tests are used.

Three types of hypothesis tests will be used in the following sections. The first test, the Wilcoxon rank sum test, compares the median values in two independent samples. The second test, the Kruskal-Wallis test, is a nonparametric analogue of the one way analysis of variance used to test for the difference in medians across more than two samples. The final test used, is the Wilcoxon sign rank test, which tests for a difference in median value for a paired sample (across time). The theory for each test is presented in the following sections.

2.4.1 The Wilcoxon Rank Sum Test

The Wilcoxon rank sum test is the non-parametric analogue of the difference of two means hypothesis test. The Wilcoxon rank sum tests for a difference in median for two



(a) Female Change in Hamilton Score

(b) Male Change in Hamilton Score

Figure 2.18: Boxplots showing the distribution of the change in Hamilton scores (after treatment - baseline) for the exploratory dataset.

independent groups. The null hypothesis is that the two medians are equal and the alternative is that they differ.

$$H_o : M_1 = M_2$$
$$H_a : M_1 \neq M_2$$

Let X denote the random variable with the smaller sample size and Y the variable with the larger sample size then the test statistic is (Gibbons, 1971):

$$W_N = \sum_{i=1}^{N} Z_i \tag{2.1}$$

where $Z_i = 1$ if the ith random variable in the combined ordered sample is an X and $Z_i = 0$ if it is a Y for the N total observations.

2.4.2 The Kruskal-Wallis Test

To investigate differences in the personality and symptom distributions across the three groups (brain study normal males and the never ill male and female relatives of bipolar patients) the Kruskal-Wallis test was used. Further details can be found in standard statistics texts such as McClave and Sincich (2000).

The null hypothesis is that the probability distributions for the i groups are identical. The alternative hypothesis is that at least two of the group's distributions differ in

location.

$$H_o : M_1 = M_2 = M_3 = \cdots = M_i$$
$$H_a : \text{at least two of the } M_i\text{'s differ}$$

The test statistic is as follows (Gibbons, 1971)

$$H = \frac{12}{n(n+1)} \sum_{j=1}^{p} \frac{R_j^2}{n_j} - 3(n+1), \tag{2.2}$$

where there are p groups; n_j is the sample size for group j; R_j is the rank sum, where the rank is calculated across all the samples, for the jth sample; and $n = n_1 + n_2 + \cdots + n_p$. The statistic follows a χ^2 distribution with $p - 1$ degrees of freedom.

2.4.3 Wilcoxon Signed Rank Test

The depressed patient datasets contain information on personality and symptoms at baseline (before treatment) and six months later (after treatment). To investigate any changes in personality and symptoms of depression, across time, the Wilcoxon signed rank test was used. The Wilcoxon signed rank test is a non-parametric analogue of the paired difference hypothesis test, the theory for this test follows. Further details can be found in most non-parametric books, a good starting point is Gibbons (1971).

The Wilcoxon signed rank test, tests for a change in trait or symptom level across all paired observations in the dataset. The null hypothesis is that there is no difference between observations before and after treatment, with the corresponding alternative hypothesis that there is a difference.

$$Ho : M_0 = M_1$$
$$H_a : M_0 \neq M_1$$

where M_0 is the median at baseline and M_1 is the median after treatment for a particular trait or symptom.

Let X denote the trait/symptom levels at baseline and Y the level at six months. Then the differences are $D_i = X_i - Y_i$ giving N pairs. It is assumed that these are independent observations from a population of differences. The differences are ranked using (Gibbons, 1971)

$$r(|D_i|) = \sum_{k=1}^{N} S(|D_i - D_k|), \tag{2.3}$$

where

$$S_u = \begin{cases} 1 & \text{if } u \geqslant 0 \\ 0 & \text{if } u < 0. \end{cases} \tag{2.4}$$

The test statistic is the smaller of either the sum of ranks of the positive differences (T^+) or the sum of the ranks of the absolute value of the negative differences (T^-), where

$$T^+ = \sum_{i=1}^{N} Z_i r(|D_i|) \tag{2.5}$$

and

$$T^- = \sum_{i=1}^{N} (1 - Z_i) r(|D_i|) \tag{2.6}$$

for

$$Z_i = \begin{cases} 1 & \text{if } D_i > 0 \\ 0 & \text{if } D_i < 0. \end{cases} \tag{2.7}$$

Matlab (Version 6.1.0 Release 12.1, The MathWorks, Inc.) was used to calculate p-values using, for large samples, a normal distribution with

$$z = \frac{4T^+ - N(N+1)}{\sqrt{2N(N+1)(2N+1)/3}}. \tag{2.8}$$

2.4.4 Comparison of the Depressed Patient Exploratory and Confirmatory Datasets

The Wilcoxon rank sum test for independent samples was used to compare the exploratory and confirmatory datasets. Table 2.6 presents the results from testing for differences between the exploratory and confirmatory groups of depressed females on each of the personality traits. Significant differences are presented in bold font. The first column presents the traits tested followed by the p-value for the hypothesis test at baseline and then after treatment. There was only one significant result at the 5% level of significance. The exploratory females were significantly different to the confirmatory females on the trait novelty seeking, at baseline. After treatment there were no significant differences.

Table 2.7 presents the Wilcoxon rank sum test results from comparing the exploratory sample of depressed males to the confirmatory depressed males on the seven TCI traits both before and after treatment. The table presents the p-values from the test and significant p-values, using the 5% level of significance, are shown in bold. There was one

Traits	Baseline	Post Treatment
Novelty Seeking	**0.035**	0.647
Harm Avoidance	0.755	0.605
Reward Dependence	0.055	0.477
Persistence	0.116	0.514
Self Directedness	0.987	0.588
Cooperativeness	0.547	0.205
Self Transcendence	0.420	0.573

Table 2.6: Comparison of the personality of the exploratory and confirmatory datasets for the depressed females.

Traits	Baseline	Post Treatment
Novelty Seeking	0.299	0.803
Harm Avoidance	0.438	0.865
Reward Dependence	0.089	0.783
Persistence	**0.011**	0.163
Self Directedness	0.378	0.793
Cooperativeness	0.879	0.582
Self Transcendence	0.150	0.077

Table 2.7: Comparison of the personality of the exploratory and confirmatory datasets for the depressed males.

Symptoms	Baseline	Post Treatment
Somatisation	0.378	0.226
Obsessive Compulsive	**0.003**	0.256
Interpersonal Sensitivity	0.309	0.949
Depression	**0.001**	0.845
Anxiety	**0.016**	0.964
Anger Hostility	**0.045**	0.435
Phobic Anxiety	0.061	0.388
Paranoid Ideation	0.745	0.355
Psychotocism	**0.015**	0.366

Table 2.8: Comparison of the symptoms of the exploratory and confirmatory datasets for the depressed females.

significant difference for the males. Persistence levels are significantly different between the exploratory and confirmatory male datasets, before treatment.

The story changes however, for the symptoms (Tables 2.8 and 2.9). After treatment there were no significant differences. At baseline however, significant differences were found across most of the symptoms. Inspection of Figures 2.12 to 2.15 shows that the distribution of the symptoms at baseline does appear similar, however the symptom levels for the confirmatory dataset are lower than those of the exploratory dataset. This suggests that the confirmatory dataset has patients who are less depressed than the original exploratory dataset. Whilst not ideal, for a confirmatory dataset, it is still appropriate to use the data in this fashion as the distribution of symptoms is similar across the groups, the underlying data collection method was the same and few differences were seen in the personality distributions across the exploratory and confirmatory groups.

2.4.5 Comparison of the Normal Datasets

The Kruskal-Wallis test was used to compare the three normal groups on each of the TCI traits. The results are presented in Table 2.10. All the p-values are significant indicating there are significant differences between at least two of the three groups for all seven TCI traits, at the 10% level of significance. In fact the first six are significant at the 5% level of significance.

As all the traits show differences, pairwise comparisons using the Wilcoxon rank sum test for independent samples were used to further investigate the differences. The results are presented in Table 2.11. These results indicate that all three data sets show various differences on certain traits. In particular the brain study volunteers show less differences

Symptoms	Baseline	Post Treatment
Somatisation	**0.035**	0.313
Obsessive Compulsive	**0.019**	0.403
Interpersonal Sensitivity	**0.004**	0.391
Depression	**0.000**	0.462
Anxiety	**0.003**	0.219
Anger Hostility	**0.001**	0.661
Phobic Anxiety	**0.038**	0.911
Paranoid Ideation	**0.031**	0.264
Psychotocism	**0.011**	0.868

Table 2.9: Comparison of the symptoms of the exploratory and confirmatory datasets for the depressed males.

Traits	p-value
Novelty Seeking	**0.003**
Harm Avoidance	**0.028**
Reward Dependence	**0.000**
Persistence	**0.000**
Self Directedness	**0.000**
Cooperativeness	**0.000**
Self Transcendence	0.051

Table 2.10: Comparison of the brain study volunteers and the never ill relatives of bipolar patients.

	Groups Compared		
Traits	Brain Males vs Never Ill Males	Brain Males vs Never Ill Females	Never Ill Males vs Never Ill Females
Novelty Seeking	**0.002**	**0.002**	0.886
Harm Avoidance	0.082	0.505	**0.012**
Reward Dependence	**0.011**	**0.000**	**0.016**
Persistence	**0.021**	0.141	**0.000**
Self Directedness	**0.000**	**0.000**	0.203
Cooperativeness	**0.000**	**0.000**	0.899
Self Transcendence	**0.013**	0.206	0.217

Table 2.11: Further comparison of the normal datasets.

with the never ill female relatives from the bipolar study than the never ill male relatives. The brain volunteers and never ill females have significantly different medians for novelty seeking, reward dependence, self directedness and self transcendence, whereas the brain volunteers and the never ill males have different medians for novelty seeking, reward dependence, persistence, self directedness, and cooperativeness. The never ill males and females have significantly different medians for harm avoidance, reward dependence and persistence.

The normal males from the brain study were expected to potentially be different on the traits novelty seeking and harm avoidance due to nature of the study. This was indeed true for novelty seeking (compared to the never ill relatives of bipolar patients) but not so for harm avoidance. Of note are the differences for the character traits. The brain volunteers are different to the never ill males on all three character traits and compared to the never ill females, are significantly different on self directedness and cooperativeness. The sample sizes are small and the never ill bipolar relatives are not randomly selected, so it is difficult to make an overall conclusion on how "normal" the brain study volunteers are.

2.4.6 Gender Differences in the Depressed Datasets

The Wilcoxon rank sum test for independent samples was used to test for differences between males and females in both depressed groups. Table 2.12 presents gender comparison results for the personality of the exploratory datasets and Table 2.13 presents the gender comparison results for the personality of the confirmatory datasets. Table 2.14 presents the gender comparison results for the symptoms of the exploratory samples of depressed males and females, and Table 2.15 presents the gender comparison results for the symptoms of the confirmatory samples of depressed males and females. The p-values for the test statistic are reported and significant p-values ($p < 0.05$) are presented in bold.

Investigation of gender differences shows that in regard to personality, there are significant differences at both time points for both datasets in the traits reward dependence and cooperativeness (Tables 2.12 and 2.13). So for both time points, before and after treatment, the only significant differences in personality between the depressed males and females, for both the exploratory and confirmatory datasets, are for the traits of reward dependence and cooperativeness. All the other traits show no significant differences across gender.

Turning to the symptom data, the exploratory dataset exhibits one symptom with a significant difference at baseline, namely paranoid ideation. At six months there are now only three symptoms that do not exhibit a significant difference in the median, these are somatisation, anger hostility and phobic anxiety. The confirmatory dataset has

Traits	Baseline	Post Treatment
Novelty Seeking	0.240	0.766
Harm Avoidance	0.135	0.890
Reward Dependence	**0.005**	**0.000**
Persistence	0.563	0.086
Self Directedness	0.272	0.130
Cooperativeness	**0.001**	**0.021**
Self Transcendence	0.296	0.068

Table 2.12: Gender comparison for the personality of the depressed patients (exploratory sample).

Traits	Baseline	Post Treatment
Novelty Seeking	0.611	0.868
Harm Avoidance	0.569	0.924
Reward Dependence	**0.000**	**0.007**
Persistence	0.259	0.700
Self Directedness	0.088	0.246
Cooperativeness	**0.000**	**0.002**
Self Transcendence	0.910	0.389

Table 2.13: Gender comparison for the personality of the depressed patients (confirmatory sample).

Symptoms	Baseline	Post Treatment
Somatisation	0.318	0.212
Obsessive Compulsive	0.113	**0.023**
Interpersonal Sensitivity	0.690	**0.014**
Depression	0.355	**0.032**
Anxiety	0.605	**0.005**
Anger Hostility	0.151	0.185
Phobic Anxiety	0.630	0.392
Paranoid Ideation	**0.047**	**0.009**
Psychotocism	0.056	**0.026**

Table 2.14: Gender comparison for the symptoms of the depressed patients (exploratory sample).

Symptoms	Baseline	Post Treatment
Somatisation	0.507	0.595
Obsessive Compulsive	0.225	**0.032**
Interpersonal Sensitivity	0.066	0.102
Depression	0.097	0.070
Anxiety	0.266	0.070
Anger Hostility	0.301	0.135
Phobic Anxiety	0.805	0.140
Paranoid Ideation	0.759	0.060
Psychotocism	0.302	**0.009**

Table 2.15: Gender comparison for the symptoms of the depressed patients (confirmatory sample).

Traits	Females	Males
Novelty Seeking	0.520	**0.029**
Harm Avoidance	**0.000**	**0.008**
Reward Dependence	**0.000**	0.236
Persistence	0.600	0.076
Self Directedness	**0.000**	**0.002**
Cooperativeness	**0.002**	**0.010**
Self Transcendence	0.134	0.190

Table 2.16: Comparison of the personality traits across time for the depressed males and females (exploratory sample).

significant gender differences in the median value of obsessive compulsive symptoms and psychotocism, after treatment only.

2.4.7 Comparison of the Traits and Symptoms Across Time

The results of the Wilcoxon sign rank test, used to compare the personality scores across time for the exploratory sample of depressed males and females are presented in Table 2.16. The table presents the p-value associated with each hypothesis test with the significance level taken at $\alpha = 0.05$. In both the depressed females and males, harm avoidance, self directedness and cooperativeness change significantly with improvement of symptoms. Persistence and self transcendence remain stable for both groups. There are gender differences in the stability or otherwise of novelty seeking and reward dependence

Traits	Females	Males
Novelty Seeking	0.322	**0.022**
Harm Avoidance	**0.000**	**0.020**
Reward Dependence	0.151	**0.031**
Persistence	**0.001**	0.092
Self Directedness	**0.000**	**0.006**
Cooperativeness	**0.000**	0.263
Self Transcendence	0.692	0.601

Table 2.17: Comparison of the personality traits across time for the depressed males and females (confirmatory sample).

Symptoms	Females	Males
Somatisation	**0.000**	**0.000**
Obsessive Compulsive	**0.000**	**0.000**
Interpersonal Sensitivity	**0.000**	**0.000**
Depression	**0.000**	**0.000**
Anxiety	**0.000**	**0.000**
Anger Hostility	**0.000**	**0.000**
Phobic Anxiety	**0.000**	**0.000**
Paranoid Ideation	**0.000**	**0.000**
Psychotocism	**0.000**	**0.000**

Table 2.18: Comparison of symptoms across time for the depressed males and females (exploratory sample).

Symptoms	Females	Males
Somatisation	**0.000**	**0.000**
Obsessive Compulsive	**0.000**	**0.000**
Interpersonal Sensitivity	**0.000**	**0.000**
Depression	**0.000**	**0.000**
Anxiety	**0.000**	**0.000**
Anger Hostility	**0.000**	**0.000**
Phobic Anxiety	**0.000**	**0.001**
Paranoid Ideation	**0.000**	**0.000**
Psychotocism,	**0.000**	**0.000**

Table 2.19: Comparison of symptoms across time for the depressed males and females (confirmatory sample).

with improvement of symptoms.

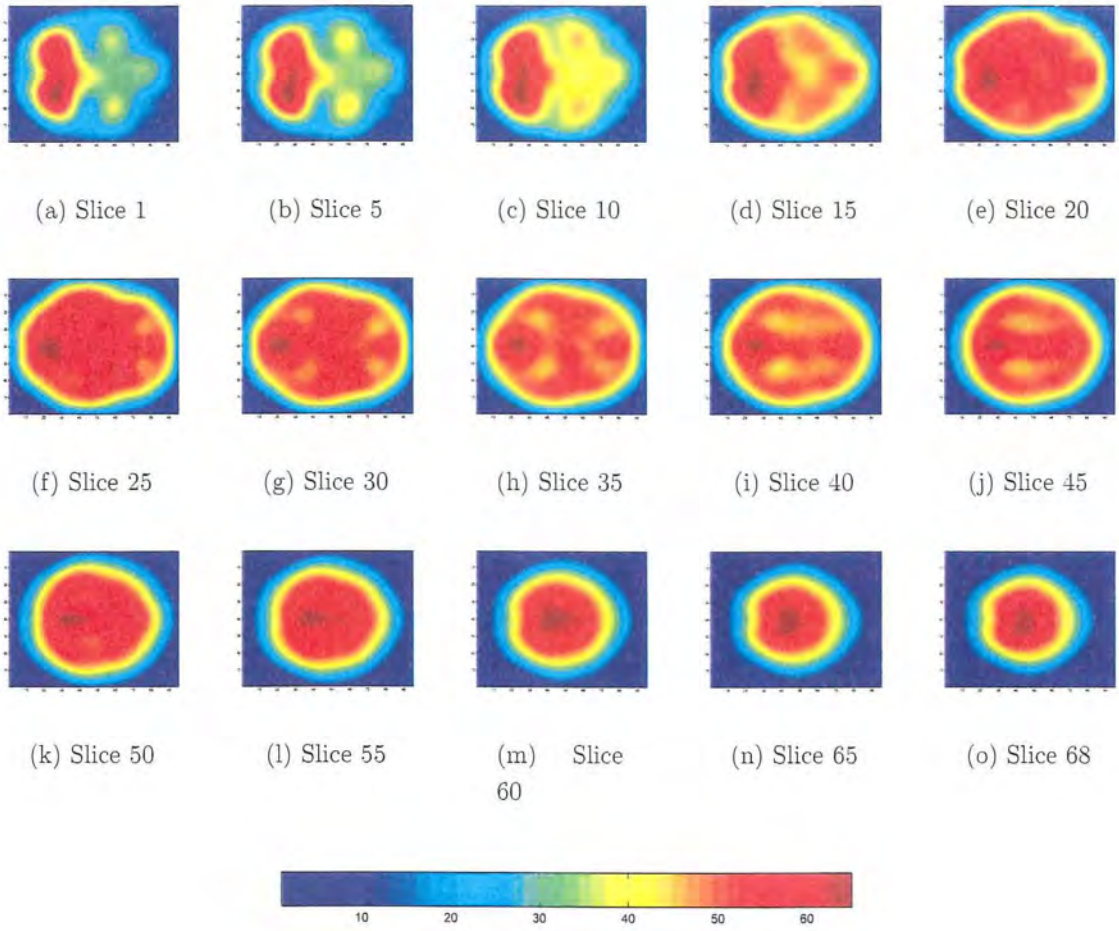
The confirmatory dataset shows significant changes across time in the personality data (Table 2.17). Harm avoidance, persistence, self directedness, and cooperativeness have changed significantly across time for the females and novelty seeking, harm avoidance, reward dependence, and self directedness have changed significantly for the males.

Table 2.18 presents the Wilcoxon signed rank test results (p-values), on the symptom scores, for the depressed males and females. Significant p-values ($p < 0.05$) are in bold. All symptoms for both males and females show significant changes across time. From the frequency distributions (Figures 2.12 to 2.15) we know that this significant change, is more specifically, a reduction in symptoms after treatment as would be expected as the patients have undergone treatment for their depression.

2.5 Brain Images

The brain images contain the counts of radiation detected in each voxel (three dimensional pixel). Figure 2.19 demonstrates the information contained in each image. Varying transverse slices have been plotted from Slice One, which is the bottom of the brain, up to Slice 68, which is the top of the brain. False colour has been used to demonstrate high flow and low flow areas using the colour bar plotted at the bottom of the figure. From this functional image the general brain structure is visible.

The brain images contain more than 500 000 voxels making any analyses difficult. The brain images are analysed in Chapter 6, during which the images are realigned and normalised into a standard brain space so that comparisons can be made of the same area



(a) Slice 1 (b) Slice 5 (c) Slice 10 (d) Slice 15 (e) Slice 20

(f) Slice 25 (g) Slice 30 (h) Slice 35 (i) Slice 40 (j) Slice 45

(k) Slice 50 (l) Slice 55 (m) Slice 60 (n) Slice 65 (o) Slice 68

(p) Colour Scale

Figure 2.19: Various transverse slices ordered from the bottom of the brain (Slice 1) to the top of the brain (Slice 68).

in each brain.

2.6 Summary

This chapter has detailed the specific data collection protocols used in this study. Basic statistical analyses have been conducted and show that in general, the data is not normal and is often highly skewed. The symptom data is often left truncated.

Non-parametric hypothesis tests were used to compare the exploratory and confirmatory depressed patient datasets. For the personality traits the females were significantly different in baseline novelty seeking and the males were significantly different in baseline persistence. Other than that the exploratory and confirmatory datasets were not significantly different on the other traits, before and after treatment. The symptom data showed more significant differences at baseline for both the males and females and none after treatment. The exploratory and confirmatory female datasets were significantly different at baseline on obsessive compulsive, depression, anxiety, anger hostility and psychotocism. The exploratory and confirmatory males were significantly different on all nine symptoms at baseline.

A similar approach was used to compare the three normal datasets. Comparison of the males from the brain study with the never ill relatives of the bipolar patients found that they were significantly different on the traits of novelty seeking, harm avoidance, self directedness and cooperativeness. In addition to these differences the males from the brain study were significantly different to the never ill male relatives of bipolar patients on persistence and self transcendence. Comparison of gender in the never ill relatives found that there were significant differences between males and females on the traits harm avoidance, reward dependence and persistence.

Investigation of the gender differences in the depressed patient exploratory and confirmatory datasets at both time points, found that males and females were significantly different on the personality traits of reward dependence and cooperativeness. No significant differences were found for novelty seeking, harm avoidance, persistence, self directedness and self transcendence. Investigation into gender differences for the depression symptoms found varying results between the confirmatory and exploratory datasets. At baseline, in the exploratory dataset, there was a significant difference between males and females for the symptom of paranoid ideation. This changed after treatment with the five symptoms of obsessive compulsive, interpersonal sensitivity, depression, anxiety and paranoid ideation showing significant differences. The confirmatory dataset had no significant gender differences at baseline and two significant differences after treatment, namely obsessive compulsive symptoms and psychotocism.

The non-parametric hypothesis tests were also used to investigate the changes in symp-

toms and personality across time. Significant differences across time were found for the exploratory females personality traits of harm avoidance, reward dependence, self directedness and cooperativeness. The confirmatory females showed significant differences across time in harm avoidance, persistence, self directedness and cooperativeness. The exploratory males showed differences to the females in that novelty seeking, harm avoidance, self directedness and cooperativeness were significantly different across time. The confirmatory males were significantly different across time in novelty seeking, harm avoidance, reward dependence and self directness. In all four groups two traits were consistently different across time, that is harm avoidance and self directedness. The symptoms would be expected to improve with treatment and thus it is no surprise to see that all symptoms were significantly different across time for all four groups.

The underlying structure and differences across groups, gender and time will be investigated further using components analysis and structural equation modelling in the next chapters. The final extension to the work investigating personality and symptoms of depression is conducted in Chapter 5, where general additive models are used to model the relationship from personality to symptoms of depression and vice versa.

Chapter 3

The Underlying Structure of Personality and Symptoms of Depression: - An Exploratory Study

This chapter uses independent component analysis, along with principal component and factor analysis, to investigate the underlying latent structure of personality and symptoms of depression. The latent structure of the data may be more informative than the manifest (observed) variables. The exploratory datasets are used to develop the component models from the principal component, independent component and factor analysis methods. Structural equation modelling in the form of confirmatory factor analysis is conducted on the confirmatory dataset, in Chapter 4 to obtain the most plausible model out of all the component models developed.

Component analysis is used in this chapter to investigate the underlying covariance structure of the TCI and SCL data and reduce the dimensionality. New principal component, independent component and factor analysis variables are developed and these may better describe any relationships between personality and symptoms of depression. These new variables are often more informative in further statistical analyses.

Independent component analysis (ICA) (Hyvärinen, 2001) greatly extends the principal component method by requiring independence of the components, rather than decorrelated components. Principal component analysis (PCA) (Hotelling, 1933) uses a transformation of variables to describe the original variance structure in a more compact manner, with little loss of information. Factor analysis (FA) (Harman, 1976) is also used in this chapter, as it is the most appropriate technique for investigating latent structures in the presence of noise. Both the PC and IC analysis methods do not allow for measurement error, whereas FA does.

This chapter introduces the theory of ICA, PCA and FA then applies these to the TCI

and SCL data of the exploratory depressed patients. This study is different to most of the studies in the literature, which investigate the factor structure of the questionnaires rather than the traits. This study had a relatively small sample size making analysis of the original questionnaire data unadvisable. The trait structure, which has been well documented in the literature, was used directly to investigate the underlying structure. Rather than looking at the questions that make up the traits, the interactions between the traits are being investigated. It is easier to model the traits, as a continuous model can then be used, rather than modelling the discrete questionnaire outcomes. Multigroup analyses are conducted first to look for common component structure across gender. The methods for determining the optimum number of components to retain give varying results. Thus for each dataset a number of IC/PC/FA structures have been found. Structural equation modelling will then be used in Chapter 4 to determine the best model on a confirmatory dataset.

3.1 Independent Component Analysis

Independent component analysis (ICA) (Jutten and Herault, 1991) is a recent development, which finds a transformation so that the data are not only decorrelated but are maximally statistically independent. In comparison PCA finds orthogonal decorrelated basis vectors. ICA has had applications in the medical field including identifying artefacts in MEG recordings (Vigário et al., 1997). ICA does not appear to have been used in the personality or depression areas whilst PCA and FA are commonly used, particularly in the area of depression. ICA is a novel approach for this type of data.

ICA developed from the blind source separation (BSS) problem which involved recovering orginal independent sources that had been mixed together. The independent sources $\mathbf{s}(t) = [s_1(t), s_2(t), ..., s_M(t)]^T$ are mixed by some matrix A to give the observed sources as follows (Roberts and Everson, 2001)

$$\mathbf{x}(t) = A\mathbf{s}(t). \tag{3.1}$$

ICA and BSS want to find a separating matrix W which recovers the estimated original sources $\mathbf{a}(t)$

$$\mathbf{a}(t) = W\mathbf{x}(t). \tag{3.2}$$

If W is found such that the statistical independence between the components of $\mathbf{a}(t)$ is maximised then these are the estimated sources (Roberts and Everson, 2001).

The General Mixing Model

Let $\mathbf{s}(t) = [s_1(t), s_2(t), \ldots, s_M(t)]^T$ be the source signals from M sources. The N-dimensional observations form the vector $\mathbf{x}(t) = [x_1(t), x_2(t), \ldots, x_N(t)]^T$ which are generated from some mixing function (Roberts and Everson, 2001)

$$\mathbf{x}(t) = \mathbf{f}(\mathbf{s}(t)) + \mathbf{n}(t), \tag{3.3}$$

where $\mathbf{n}(t)$ is observational noise and the function $\mathbf{f} : \mathbb{R}^M \to \mathbb{R}^N$ is unknown.

Traditional ICA assumes that the sources are mixed linearly by a matrix $A \in \mathbb{R}^{N \times M}$. Thus the mixing model reduces to (Roberts and Everson, 2001)

$$\mathbf{x}(t) = A\mathbf{s}(t) + \mathbf{n}(t). \tag{3.4}$$

The mixing model can be further reduced by assuming that there is no noise. The majority of traditional ICA models make this assumption leading to

$$\mathbf{x}(t) = A\mathbf{s}(t). \tag{3.5}$$

Independent Sources

ICA models assume that the original sources are independent. Thus the factorisation of the joint source density function is (Roberts and Everson, 2001)

$$p(\mathbf{s}) = \prod_{m=1}^{M} p(s_m(t)). \tag{3.6}$$

This factorisation can be used to check for independence. If the probability density function of the estimated sources factorises then the recovered sources are independent. Mutual information is used to measure the independence between the recovered sources and depends on the probability density function of $\mathbf{x}$ rather than $\mathbf{x}$ itself. The mutual information can be defined in terms of the differential entropy (Roberts and Everson, 2001)

$$H[\mathbf{x}] = H[p(\mathbf{x})] \equiv -\int p(\mathbf{x}) \log p(\mathbf{x}) d\mathbf{x}, \tag{3.7}$$

where entropy is a measure of the average amount of information that can be gained from observing, in this case, $\mathbf{x}$. Entropy, a concept originally from statistical physics, forms a cornerstone of information theory. Differential entropy is used for continuous variables rather than ordinary entropy.

Negentropy is another useful statistical tool from information theory. It measures the non-Gaussianity of a random variable ($\mathbf{x}$) by comparing the entropy of the random variable

to that of a Gaussian random variable with the same covariance matrix. Negentropy is thus zero if and only if $\mathbf{x}$ is Gaussian (Cover and Thomas, 1991).
This can be extended to the joint entropy of two random variables $\mathbf{x}$ and $\mathbf{y}$ using (Roberts and Everson, 2001)

$$H[\mathbf{x},\mathbf{y}] = -\int p(\mathbf{x},\mathbf{y}) \log p(\mathbf{x},\mathbf{y}) d\mathbf{x} d\mathbf{y}. \tag{3.8}$$

Also the entropy of x given y

$$H[\mathbf{x}|\mathbf{y}] = -\int p(\mathbf{x},\mathbf{y}) \log p(\mathbf{x}|\mathbf{y}) d\mathbf{x} d\mathbf{y}, \tag{3.9}$$

so that

$$\begin{aligned} H[\mathbf{x},\mathbf{y}] &= H[\mathbf{x}] + H[\mathbf{y}|\mathbf{x}] && (3.10)\\ &= H[\mathbf{y}] + H[\mathbf{x}|\mathbf{y}]. && (3.11) \end{aligned}$$

Thus the mutual information between two variables can be defined in terms of their entropies as (Roberts and Everson, 2001)

$$\begin{aligned} I[\mathbf{x};\mathbf{y}] &= H[\mathbf{x}] + H[\mathbf{y}] - H[\mathbf{x},\mathbf{y}] && (3.12)\\ &= H[\mathbf{x}] - H[\mathbf{x}|\mathbf{y}] && (3.13)\\ &= H[\mathbf{y}] - H[\mathbf{y}|\mathbf{x}]. && (3.14) \end{aligned}$$

The mutual information is the difference in information between observing $\mathbf{x}$ and $\mathbf{y}$ separately and observing $\mathbf{x}$ and $\mathbf{y}$ jointly. It is zero when $\mathbf{x}$ and $\mathbf{y}$ are independent. The mutual information between the components of $\mathbf{a}$ is (Roberts and Everson, 2001)

$$\begin{aligned} I[\mathbf{a}] &\equiv I[\mathbf{a};\{a_m\}] && (3.15)\\ &= \sum_{m=1}^{M} H[a_m] - H[\mathbf{a}] && (3.16)\\ &= \int p(\mathbf{a}) \log \frac{p(\mathbf{a})}{\prod_{m=1}^{M} p_m(a_m)} d\mathbf{a}. && (3.17) \end{aligned}$$

If $I[\mathbf{a}] = 0$ then the estimated sources do not have common information and independence of the sources has been achieved. The mutual information between the recovered sources is the same as the Kullback-Leibler divergence between the two probability density functions (Roberts and Everson, 2001). In the case of linear mixing ICA finds the separating matrix that minimises the Kullback-Leibler divergence.

Ambiguities from Scaling and Permutation

Equation 3.4 has a fundamental ambiguity in scaling of the estimated sources (Roberts and Everson, 2001). Roberts and Everson (2001) show that this is due to the fact that a scale change in the source by a factor λ is compensated for by a division of λ in the mixing matrix for the corresponding column. Likewise the mutual information does not depend on the scale of the estimated sources ($I[\mathbf{a}] = I[D\mathbf{a}]$ for some nonzero diagonal matrix D). The order of the components of $\mathbf{a}$ makes no difference to the independence of these components so that $I[\mathbf{a}] = I[P\mathbf{a}]$ when P is a permutation matrix. So the original sources can only be estimated within an arbitrary scaling and permutation.

For the situation of zero noise the mutual information is (Roberts and Everson, 2001)

$$I[\mathbf{s}] = I[W\mathbf{s}] \tag{3.18}$$

$$= I[PDW\mathbf{s}]. \tag{3.19}$$

ICA using a Square Mixing Matrix and without Noise

In the absence of a noise term ICA, like PCA, is not a true generative model (Roberts and Everson, 2001) thus a psuedo-likelihood is calculated rather than a true likelihood. The likelihood of a single observation for ICA with square mixing and without noise is given by (Roberts and Everson, 2001)

$$p(\mathbf{x}|A, \mathbf{s}) = \frac{1}{|\det A|} \prod_{m=1}^{M} p(s_m). \tag{3.20}$$

Rewriting this in terms of the separating matrix W (Equation 3.2) leads to (Roberts and Everson, 2001)

$$\log l(\mathbf{x}) = \log p(\mathbf{x}|W, \mathbf{a}) = \log |\det W| + \sum_{m=1}^{M} \log p(\mathbf{a}_m). \tag{3.21}$$

For the m^{th} estimated source the estimated entropy is

$$H_m[a_m] = -\frac{1}{T} \sum_{t=1}^{T} \log p(a_m(t)) \tag{3.22}$$

$$\approx -\int p(a_m) \log p(a_m) da_m. \tag{3.23}$$

The log likelihood for a group of T observations is thus (Roberts and Everson, 2001)

$$\mathcal{L} = \log|\det W| + \frac{1}{T}\sum_{t=1}^{T}\sum_{m=1}^{M}\log p(\mathbf{a}_m(t)) \tag{3.24}$$

$$= \log|\det W| + \frac{1}{T}\sum_{t=1}^{T}\sum_{m=1}^{M}\log p(\sum_j W_{mj}\mathbf{x}_j(t)) \tag{3.25}$$

$$= \log|\det W| - \sum_{m=1}^{M} H_m[a_m]. \tag{3.26}$$

Equation 3.25 can be differentiated and solved for the maximum likelihood separating function. Many of the ICA alorithms use this as the basis for the source separation. This maximum likelihood separating matrix gives recovered sources that are the most independent (MacKay, 1996; Cardoso, 1997), this is demonstrated below.

$$I[\mathbf{a}] = \int p(\mathbf{a})\log\frac{p(\mathbf{a})}{\prod_{m=1}^{M}p(a_m)}d\mathbf{a} \tag{3.27}$$

$$= \int p(\mathbf{a})\log p(\mathbf{a})d\mathbf{a} + \sum_{m=1}^{M} H_m[a_m] \tag{3.28}$$

$$= -\log|\det W| + H[\mathbf{x}] + \sum_{m=1}^{M} H_m[a_m], \tag{3.29}$$

Comparing this to equation 3.26 gives

$$I[\mathbf{a}] = H[\mathbf{x}] - \mathcal{L}. \tag{3.30}$$

Thus maximisation of the likelihood is equivalent to minimisation of the mutual information as $H[\mathbf{x}]$ is constant.

Fixed Source Models

When using ICA with linear mixing, prior assumptions about the probability density functions must be made (Roberts and Everson, 2001). The basic choice of source function is one that is fixed with no adjustable parameters. The source function is often writen interms of a nonlinear function ϕ_m and a common choice for this function is

$$\phi_m(a_j) = -\frac{\partial \log p(a_m)}{\partial a_m} = \tanh(a_j). \tag{3.31}$$

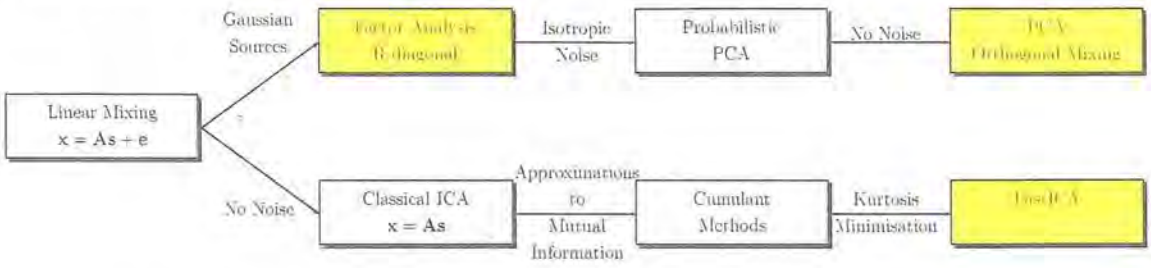


Figure 3.1: Family of ICA models (Roberts and Everson, 2001).

3.1.1 Fast ICA

FastICA (www.cis.hut.fi/projects/ica/fastica/) was used for the ICA analysis. The theory behind FastICA is published in Hyvärinen (2001, 1999a,b, 1998, 1997b,a), and Hyvärinen and Oja (1997, 2000). FastICA, FA and PCA are part of the broader hierarchy of ICA models (see Page 7 Roberts and Everson (2001)); a schema depicting this, in part, is presented in Figure 3.1 to indicate the relationships between ICA, PCA and FA. Both Fast ICA and PCA do not allow for a noise term but factor analysis does. PCA has orthogonal mixing whilst FastICA uses negentropy (normalised differential entropy) minimisation to estimate independence. Further discussion on the similarities and differences between these three methods is presented in Section 3.4.

An important pre-processing step to enable simplification of the ICA algorithms is the data transformation, known as whitening (Hyvärinen, 2001), to a new set of uncorrelated components, which have variances equal to one. Fast ICA uses the eigenvalue decomposition of the covariance matrix to do this. Let V be the matrix of eigenvectors and D the eigenvalues (diagonal matrix), then the whitening is given by (Hyvärinen, 2001):

$$\widetilde{\boldsymbol{x}} = VD^{-\frac{1}{2}}V^T\mathbf{x} \tag{3.32}$$

giving a new white matrix $\widetilde{x}$. This leads to a covariance matrix

$$E(\mathbf{x}\mathbf{x}^T) = \mathbf{I}, \tag{3.33}$$

where $\mathbf{I}$ is the identity matrix.

Hyvärinen (2001) shows that if the non-Gaussianity of $\mathbf{W}^T\mathbf{x}$ is maximised, the independent components are obtained. Fast ICA uses negentropy to measure non-Gaussianity. Entropy is a concept from information theory describing the information gained by measurement of a variable. Gaussian variables have the largest entropy. For a continuous random variable ($\mathbf{x}$) the entropy (H) is defined in equation 3.7.
Negentropy (J) is defined as the normalised differential entropy

$$J(\mathbf{x}) = H(\mathbf{x}_G) - H(\mathbf{x}), \tag{3.34}$$

for an $\mathbf{x}_G$, which has an equivalent covariance matrix to $\mathbf{x}$ but is a Gaussian random variable. In practice estimating negentropy from this equation is difficult so Fast ICA uses the following approximation (Hyvärinen, 2001)

$$J(x) \propto [E(G(x)) - E(G(\nu))]^2, \tag{3.35}$$

where $E(x)$ is the covariance matrix, ν is a standardised Gaussian variable and G is a function with the derivative

$$g(u) = \tanh(a_1 u), \tag{3.36}$$

for constant a_1. This is the formulation for the Fast ICA algorithm with robust non-linearity g.

The Fixed Point Algorithm

FastICA uses a fixed point algorithm to maximise the non-Gaussianity of $\mathbf{W}^T\mathbf{x}$. The method is an approximate Newton method. First Hyvärinen (2001) defines $\mathbf{w}$ as one component of the W matrix. The fixed points are the optima of $E[G(\mathbf{w}^T\mathbf{x})]$ where

$$F = E[\mathbf{x}g(\mathbf{w}^T\mathbf{x})] + \beta\mathbf{w} = 0, \tag{3.37}$$

using the constraint $E[(\mathbf{w}^T\mathbf{x})^2] = \|\mathbf{w}\|^2 = 1$ and β is a constant. The Jacobian of this equation is

$$JF(\mathbf{w}) = E[\mathbf{x}\mathbf{x}^T g'(\mathbf{w}^T\mathbf{x})] + \beta I. \tag{3.38}$$

For computational purposes the following simplification is used

$$E[\mathbf{x}\mathbf{x}^T g'(\mathbf{w}^T\mathbf{x})] \approx E[\mathbf{x}\mathbf{x}^T]E[g'(\mathbf{w}^T\mathbf{x})] = E[g'(\mathbf{w}^T\mathbf{x})]I, \tag{3.39}$$

and this leads to the approximate Newton iteration

$$\mathbf{w} \leftarrow \mathbf{w} - \frac{E[\mathbf{x}g(\mathbf{w}^T\mathbf{x})] + \beta\mathbf{w}}{E[g'(\mathbf{w}^T\mathbf{x})] + \beta}. \tag{3.40}$$

With simplification this leads to the basic fixed-point iteration in Fast ICA of

$$\mathbf{w} \leftarrow E[\mathbf{x}g(\mathbf{w}^T\mathbf{x})] - E[g'(\mathbf{w}^T\mathbf{x})]\mathbf{w}. \tag{3.41}$$

Further details of the iterative procedure are given in Hyvärinen (2001) and are reproduced for convenience in Table 3.1. Thus the Fast ICA algorithm iteratively finds the weight matrix W that maximises the independence of the estimated sources. This weight matrix represents the loadings of the observed variables onto each component. Independent

(i)	Whiten the data to give $\mathbf{x}$
(ii)	Choose m, the number of ICs to estimate. Set counter $p \leftarrow 1$.
(iii)	Choose an initial value of unit norm for $\mathbf{w}_p$, e.g. randomly.
(iv)	Let $\mathbf{w}_p \leftarrow E[\mathbf{x}g(\mathbf{w}_p^T\mathbf{x})] - E[g'(\mathbf{w}_p^T\mathbf{x})]\mathbf{w}$, where g is defined as equation 3.36.
(v)	Do the orthogonalisation $\mathbf{w}_p \leftarrow \mathbf{w}_p - \sum_{j=1}^{p-1}(\mathbf{w}_p^T\mathbf{w}_j)\mathbf{w}_j$.
(vi)	Let $\mathbf{w}_p \leftarrow \mathbf{w}_p / \parallel \mathbf{w}_p \parallel$.
(vii)	If $\mathbf{w}_p$ has not converged, go back to step (iv).
(viii)	Set $p \leftarrow p+1$. If $p \leq m$, go back to step (iii).

Table 3.1: ICA fixed-point algorithm (Hyvärinen, 2001)

component analysis forms a family of models, with PCA and FA part of the family. As PCA and FA are the standard approaches used in the literature these methods will also be implemented on the personality and symptom data.

An important issue in ICA analysis is the assumption of independence of the latent underlying factors. Shimizu and Kano (2003) investigate the independence of the components found in ICA. This question is important for the interpretation of components in the psychometric field. The article initiates the need for test statistics for independence of the components and suggests some statistics that may be useful for this. This developing area will be used in my future work to investigate the independence of the latent factors found in this thesis.

3.2 Principal Component Analysis

Principal component analysis (PCA) is used to reduce the number of variables, with as little loss of information as possible, to a new set of uncorrelated variables. A variable transformation is used to achieve the variable reduction. The transformed variables are the principal components (PCs). The PCs are ordered by importance, which is defined as the amount of variance explained by each PC. The key to principal component analysis is to find a new set of uncorrelated variables. The principal components turn out to be the eigenvectors of the data with the corresponding eigenvalues defining the amount of variance explained by each eigenvector.

Detailed theory on PCA can be found in many multivariate books including Manly (1986). The theory here is from Jolliffe (1986) and the equations are reproduced for convenience.

Let $\boldsymbol{x}$ be a vector of p random variables and $\boldsymbol{\Sigma}$ the covariance matrix. PCA finds a linear function $(\boldsymbol{\alpha}_1'\boldsymbol{x})$ of these variables that maximises the variance

$$var[\boldsymbol{\alpha}_1'\boldsymbol{x}] = \boldsymbol{\alpha}_1'\boldsymbol{\Sigma}\boldsymbol{\alpha}_1. \tag{3.42}$$

For a solution to be obtained from Equation 3.42 a constraint is required. The general constraint used is $\boldsymbol{\alpha}_1'\boldsymbol{\alpha}_1 = 1$. Using Lagrange multipliers (λ), Equation 3.43 is maximised by differentiating with respect to α_1' and equating to zero (see Equation 3.44): i.e.

$$var[\boldsymbol{\alpha}_1'\boldsymbol{x}] = \boldsymbol{\alpha}_1'\boldsymbol{\Sigma}\boldsymbol{\alpha}_1 - \lambda(\boldsymbol{\alpha}_1'\boldsymbol{\alpha}_1 - 1) \tag{3.43}$$

$$\boldsymbol{\Sigma}\boldsymbol{\alpha}_1 - \lambda\boldsymbol{\alpha}_1 = 0. \tag{3.44}$$

Rearranging and using $\boldsymbol{I_p}$ = identity matrix leads to,

$$(\boldsymbol{\Sigma} - \lambda\boldsymbol{I_p})\boldsymbol{\alpha}_1 = 0.$$

Clearly λ is an eigenvalue of $\boldsymbol{\Sigma}$ and $\boldsymbol{\alpha}_1$ is the corresponding eigenvector. There will be p eigenvalues and p eigenvectors. The eigenvalue/eigenvector pair that gives the maximum variance is calculated as follows:

$$\begin{aligned} var[\boldsymbol{\alpha}_1'\boldsymbol{x}] &= \boldsymbol{\alpha}_1'\boldsymbol{\Sigma}\boldsymbol{\alpha}_1 && (3.45)\\ &= \boldsymbol{\alpha}_1'\lambda\boldsymbol{\alpha}_1 \quad \text{(from Equation 3.44)} && (3.46)\\ &= \lambda\boldsymbol{\alpha}_1'\boldsymbol{\alpha}_1 && (3.47)\\ &= \lambda. && (3.48) \end{aligned}$$

Therefore the largest eigenvalue (λ) will give the maximum variance and the variance is equal to this eigenvalue. Generally the k*th* PC is $\boldsymbol{\alpha}_k'\boldsymbol{x}$ and its associated variance is λ_k. The proof that the principal components are uncorrelated can be found in Jolliffe (1986), Page 4.

The principal component scores (the observations measured on the new variables) are denoted as $\mathbf{Z}$ and can be calculated by:

$$\boldsymbol{Z} = \boldsymbol{A}'\boldsymbol{x}^*,$$

where $\boldsymbol{A}$ is a matrix with columns that are the eigenvectors of $\boldsymbol{\Sigma}$, and $\boldsymbol{x}^*$ is the matrix of standardised variables. So the first principal component is given by

$$\boldsymbol{Z}_1 = \alpha_{11}\boldsymbol{x}_1 + \alpha_{12}\boldsymbol{x}_2 + \cdots + \alpha_{1p}\boldsymbol{x}_p, \tag{3.49}$$

where $\boldsymbol{Z}_1$ is the new variable PC score for the observations. The α coefficients (loadings) are used to interpret each principal component.

3.3 Factor Analysis

Factor analysis (Harman, 1976) is fundamentally different from principal components analysis as, FA uses a specific type of model to describe the data. The model is specified as follows, for a prespecified number of factors

$$\mathbf{x} = \mathbf{\Lambda} f + \mathbf{e}, \tag{3.50}$$

where $\mathbf{\Lambda}$ is the matrix of factor loadings on the $\mathbf{f}$ factors and $\mathbf{e}$ are the error terms. Model assumptions are as follows. First the error terms are uncorrelated with each other and second they are uncorrelated with the factors. The factors are orthogonal unless an oblique rotation is used. Rotation of principal components and factors is discussed in section 3.5. For this thesis only orthogonal rotation is used.

If we let $E[\mathbf{e}\mathbf{e}'] = \mathbf{\Phi}$ then taking the covariance of both sides of equation 3.50 leads to

$$\mathbf{\Sigma} = \mathbf{\Lambda}\mathbf{\Lambda}' + \mathbf{\Phi}, \tag{3.51}$$

where $\mathbf{\Sigma}$ is the population covariance matrix estimated by the sample covariance matrix S.

This study employs maximum likelihood factor estimation to calculate $\mathbf{\Lambda}$ and $\mathbf{\Phi}$. Maximum likelihood factor estimation assumes that the data is multivariate normal. Maximum likelihood estimation finds a $\mathbf{\Lambda}$ and $\mathbf{\Phi}$ that maximise the likelihood function (Harman, 1976)

$$\log L = -\frac{N-1}{2}(\log|\mathbf{\Sigma}| + \sum_{j,k=1}^{n} \sigma^{jk} s_{jk}) + \text{function independent of } \mathbf{\Sigma}. \tag{3.52}$$

where σ^{jk} are the elements of the inverse population covariance matirx, s_{jk} are the elements of the sample covariance matrix and there are N independent observations. Further details of maximum likelihood estimation can be found in Harman (1976).

3.4 Comparison of the Component Methods

Factor analysis and principal component analysis have some fundamental differences. Factor analysis has a definite underlying model whereas principal component analysis has no such underlying model. Factor analysis concentrates on explaining the off-diagonal elements in the covariance matrix, in contrast principal component analysis concentrates on explaining the diagonal elements in the covariance matrix (Jolliffe, 2002). Factor analysis allows for an error term and this leads to uncertainty in the calculation of the

factor scores. The principal component scores can be calculated exactly but the factor scores need to be estimated. Further details on the differences between factor analysis and principal component analysis can be found in Jolliffe (2002).

Independent component anlaysis is a very recent developement in this area and originates from the blind source separation problem. Independent component analysis is related to both PCA and FA. PCA assumes Gaussian sources and no observational noise.

$$\mathbf{x}(t) = A\mathbf{s}(t). \tag{3.53}$$

The PCs are calculated by finding the direction of maximum variance and these directions turn out to be the eigenvectors. The ambiguity in the process is resolved in PCA by making the sources have unit variance ($\mathbf{s} \sim \mathcal{N}(0, I)$). ICA in the Fast ICA package is similar in that no observational noise is allowed for in the calculation, however the components are found that give maximum independence between the recovered sources. The method used to do this is fundamentally different and achieves more than the PCA method.

FA allows for observational noise in the model and the factors in the model are estimated iteratively. The FA model is

$$\mathbf{x}(t) = A\mathbf{s}(t) + \mathbf{n}(t), \qquad \mathbf{n}(t) \sim \mathcal{N}(0, R_n). \tag{3.54}$$

When the observational noise is zero, the mixing matrix square and the source priors are non-Gaussian, the model reduces to that of ICA (Roberts and Everson, 2001).

Kano et al. (2003) investigate the relationship between ICA and FA. They show that under certain conditions, when there is excess kurtosis in every blind signal, FA can be used to produce ICA results and that ICA under certain conditions, varimax-based ICA, can be used to produce the FA results. Interestingly they show that ICA does not necessarily subsume FA, in certain contexts (combination of super-Gaussian and sub-Gaussian components) ICA can not analyse the data sets. This is demonstrated in the thesis, ICA was the best method in a majority of cases but not all cases.

3.5 Rotation of the Principal Components and the Factors

If an orthogonal matrix, say $\boldsymbol{T}$, is multiplied by the factor loadings then the following indeterminacy occurs,

$$\Lambda T(\Lambda T)' = \Lambda TT'\Lambda' \tag{3.55}$$
$$= \Lambda\Lambda'. \tag{3.56}$$

Rotation of the factor loadings is used to account for this indeterminacy problem, both in factor analysis and in principal component analysis. ICA does not have this rotational ambuigity so the rotation method has not been used on the ICA loadings.

3.5.1 Varimax Rotation

The method of rotation used in this study is Varimax rotation developed by Kaiser (1958). The idea behind Varimax rotation is to achieve factors that are simple and interpretable. Maximisation of the squared variance of the loadings tends to give components that are either close to zero or one. This makes the factor easier to interpret and gives a simpler structure. The communalities of the loadings are taken into account in the equation otherwise the variables with larger communalities would contribute more to the factor rotation resulting in bias Harman (1976). Varimax rotation finds a transformation matrix that maximises the sum of the variance of the squares of the scaled loadings given by Equation 3.57

$$V = \frac{1}{p}\sum_{j=1}^{m}\left[\sum_{i=1}^{p}\widetilde{l}_{ij}^{*4} - \left(\sum_{i=1}^{p}\widetilde{l}_{ij}^{*2}\right)^2/p\right], \tag{3.57}$$

where

$\widetilde{l}_{ij}^{*} = \widehat{l}_{ij}^{*}/\widehat{h}_i$
$\widehat{l}_{ij}^{*}$ = estimated factor loadings
$\widehat{h}_i$ = communalities (sum of the squares of the loadings)
p = number of variables
m = number of PC's rotated.

Further details of the Varimax method can be found in Harman (1976). Varimax rotation retains the decorrelated orthogonal structure of the components and leads to reproducibility of the component structure.

3.6 Choosing the Number of Components to Retain or Rotate

There are a number of techniques for determining the number of components (p) to retain. They range from subjective techniques, such as finding the most interpretable components, to objective techniques such as Velicer's MAP (Velicer, 1976). A range of these techniques will be used in this study. If there is lack of agreement between techniques, all the numbers of components suggested by the methods will be used and the resultant models will be tested in Chapter 4 using confirmatory factor analysis.

3.6.1 Comparison of the Methods

Zwick and Velicer (1986) compared five different methods for determining the number of factors to rotate. The five tests were Horn's parallel analysis (PA) (Horn, 1965), Velicer's MAP (Velicer, 1976), Cattell's Scree test (Cattell, 1966), Bartlett's chi-square test (Bartlett, 1950, 1951) and Kaiser's eigenvalue greater than one rule (K1) (Kaiser, 1960). They found that K1 and Bartlett's chi-square test over estimate the number of eigenvalues to retain, and Bartlett's test was the most variable and susceptible to sample size. The Scree test was an improvement on the previous two, but had the problem of reliability from experimenter bias. Zwick and Velicer (1986) recommended the use of MAP and PA, noting that MAP tended to underestimate the number of components in situations where the test failed. The authors summarise by saying

> There is no evidence supporting the continued use of K1 or the Bartlett test as exclusive, primary methods to determine the number of major components to retain. These methods should not be used ...either PA or MAP is the method of choice, with many situations arising in which both should be used.

With this in mind the techniques of MAP, Auto Scree, Armor's Theta (Armor, 1974) and the percentage variance accounted for (PVAF) (Jolliffe, 1986) will be used.

3.6.2 The AutoScree Test

The scree test was developed by Cattell (1966). The scree plot is the term used to describe the plot of the eigenvalues of the sample covariance matrix plotted in descending order. The scree test looks for the point where the difference between successive eigenvalues is small and constant for successive values. In graphical terms this is finding a break point in the scree plot where the slope reduces significantly. This method is subjective as it generally involves the user visually finding breakpoints in the scree plot. Barrett and Kline (1982) automated the process (available in PSPWIN (www.pbarrett.net)). The

program, known as AutoScree finds up to four significant break points in the scree plot automatically to avoid user bias.

Initially AutoScree starts at the lowest eigenvalues and a least squares regression line, a tangent of slope about the regression line and 1 minus the coefficient of determination are calculated for n eigenvalues. The next eigenvalue is added to the calculation and the difference between the tangents and the difference in error are calculated. The n eigenvalues are grouped as a scree line if either the difference between the tangents or errors are larger than some prespecified value. The process is repeated from where the algorithm stopped. The final break points are those angle deviations between scree sets that are above a set value.

3.6.3 Velicer's MAP

Velicer (1976) proposed an exact stopping point for the number of components to rotate based on the matrix of partial correlations. The full details of this theory can be found in Velicer (1976), the equations are reproduced here for convenience. The procedure starts by partitioning the variables into p and m variables giving a covariance matrix as follows:

$$C = \begin{bmatrix} C_{11} & C_{12} \\ C'_{12} & C_{22} \end{bmatrix} \tag{3.58}$$

where C_{11} is size $p \times p$, C_{12} is size $p \times m$ and C_{22} is size $m \times m$. Using this partitioned formulation the matrix of partial correlations is calculated by:

$$R^*_{11} = D^{-\frac{1}{2}}(C_{11} - C_{12}C_{22}^{-1}C'_{12})D^{-\frac{1}{2}} \tag{3.59}$$

where $D = \text{diag}(C_{11} - C_{12}C_{22}^{-1}C'_{12})$. A summary statistic is calculated from the partial correlations which measures the average of the squared partial correlations after partitioning out the first m components. The stopping criterion is the mth value that gives a minimum summary statistic, f_m, where

$$f_m = \sum\sum_{i \neq j}(r^*_{ij})^2/(p(p-1)). \tag{3.60}$$

Note that r^*_{ij} is the $(i,j)th$ element of R^*_{11} in Equation 3.59. Velicer (1976) explains that f_m will decrease for the common components and increase for the unique components. Common components account for the covariance between the variables (off diagonal terms of the covariance matrix) and unique components account for the variance of the variables including the error (diagonal terms of the covariance matrix) (Harman, 1976).

3.6.4 Armor's Theta

Developed by Armor (1974), Armor's Theta measures the reliability of the k*th* set of rotated principal component scores. Let p be the number of variables, λ_h the eigenvalues and ϕ_{hk} the hk*th* element in the transformation matrix mapping the original principal component loadings onto the rotated loadings. Armor's Theta is:

$$\theta_k^* = [p/(p-1)]\left(1 - \sum_{h=1}^{m} \phi_{hk}^2/\lambda_h\right). \tag{3.61}$$

From Barrett's PSPWIN program help (www.pbarrett.net) we desire theta to be bigger than 0.5.

3.6.5 Percentage Variance Accounted For

The last method used in this study, calculates the percentage of the variance that each eigenvalue accounts for (Jolliffe, 1986). Equation 3.48 showed that each eigenvalue was equivalent to the variance of each $\boldsymbol{\alpha}'\boldsymbol{x}$ so

$$\text{Percentage Variance} = \frac{\lambda_i}{\sum_{j=1}^{p} \lambda_j} \times 100\%, \tag{3.62}$$

is the percentage variance that the ith eigenvalue accounts for. This study has chosen the number of components so that at least 90% of the variance is retained.

3.7 Testing for Common Structure Across Groups - The Flury Test

The personality and symptom study investigates two groups, the depressed males and depressed females. The same TCI and SCL variables are measured across these groups and there may be a similar component structure. To test for common principal components the chi-squared test developed by Flury (1984) can be used. It tests the null hypothesis of common principal components across k groups ($k \geq 2$) for the $p \times p$ covariance matrices ($\boldsymbol{\Sigma}$) for each group. The null hypothesis can be written as (Flury, 1984)

$$H_{CPC}: \ \boldsymbol{\Sigma_i} = \boldsymbol{\beta}\boldsymbol{\Lambda_i}\boldsymbol{\beta}', \ i = 1, \ldots, k \tag{3.63}$$

where $\boldsymbol{\beta}$ is the matrix of eigenvalues representing the common principal components (CPCs). Flury (1984) shows that the common likelihood function of $\boldsymbol{\Sigma}_1, \ldots, \boldsymbol{\Sigma}_k$ given $\mathbf{S}_1, \ldots, \mathbf{S}_k$, the sample covariance matrix for each group, for some constant (C) independent of $\boldsymbol{\Sigma}_i$ is

$$\mathcal{L}(\Sigma_1, \ldots, \Sigma_k) = C \times \prod_{i=1}^{k} e^{\mathrm{tr}(-\frac{n_i}{2}\Sigma_i^{-1}\mathbf{S}_i)} |\Sigma_i|^{-n_i/2}. \tag{3.64}$$

Computationally the likelihood function is maximised by minimising the following function

$$g(\Sigma_1, \ldots, \Sigma_k) = -2 \log \mathcal{L}(\Sigma_1, \ldots, \Sigma_k) + 2 \log C \tag{3.65}$$

$$= \sum_{i=1}^{k} n_i (\log |\Sigma_i| + \mathrm{tr}\Sigma_i^{-1}\mathbf{S}_i). \tag{3.66}$$

Under the null hypothesis and for $\mathbf{\Lambda}_i$, a group specific diagonal matrix of eigenvalues (λ_{ij}), then (Flury, 1984)

$$\log |\Sigma_i| = \sum_{j=1}^{p} \log \lambda_{ij}, \tag{3.67}$$

for $i = 1, \ldots, k$, and

$$\mathrm{tr}\Sigma_i^{-1}\mathbf{S}_i = \mathrm{tr}(\boldsymbol{\beta}\mathbf{\Lambda}_i^{-1}\boldsymbol{\beta}'\mathbf{S}_i) \tag{3.68}$$

$$= \sum_{j=1}^{p} \boldsymbol{\beta}_j'\mathbf{S}_i\boldsymbol{\beta}_j/\lambda_{ij}, \tag{3.69}$$

note that the jth column of $\boldsymbol{\beta}$ is $\boldsymbol{\beta}_j$. Using the above information the minimisation function becomes

$$g(\Sigma_1, \ldots, \Sigma_k) = \sum_{i=1}^{k} n_i [\sum_{j=1}^{p} (\log \lambda_{ij} + \boldsymbol{\beta}_j'\mathbf{S}_i\boldsymbol{\beta}_j/\lambda_{ij}]. \tag{3.70}$$

The minimisation of this leads to the following equation (Flury, 1984)

$$\boldsymbol{\beta}_l'(\sum_{i=1}^{k} n_i \frac{\lambda_{il} - \lambda_{ij}}{\lambda_{il}\lambda_{ij}}\mathbf{S}_i)\boldsymbol{\beta}_j = 0, \tag{3.71}$$

for $l, j = 1, \ldots, p$ and $l \neq j$. This system of equations is solved under the orthonormality conditions of $\boldsymbol{\beta}'\boldsymbol{\beta} = \boldsymbol{I}_p$ using the FG algorthim developed by (Flury and Gautschi, 1986). This solution leads to the maximum likelihood estimates $\widehat{\boldsymbol{\beta}}$ and $\widehat{\lambda_{ij}}$. The likelihood function from equation 3.64 becomes

$$\mathcal{L}(\widehat{\Sigma}_1, \ldots, \widehat{\Sigma}_k) = C \prod_{i=1}^{k} e^{-pn_i/2} |\widehat{\Sigma}_i|^{-n_i/2}. \tag{3.72}$$

	Chi-Square	df	p-value
TCI at baseline	30.00	21	0.09
TCI after treatment	29.08	21	0.11
SCL at baseline	37.18	36	0.41
SCL after treatment	79.76	36	0.00

Table 3.2: Flury chi-squared test results for the comparison of the exploratory depressed males and females.

This leads to a log likelihood ratio test statistic for the null hypothesis of common principal components of (Flury, 1984)

$$\chi^2 = -2 \log \frac{\mathcal{L}(\widehat{\boldsymbol{\Sigma}}_1, \ldots, \widehat{\boldsymbol{\Sigma}}_k)}{\mathcal{L}(\mathbf{S}_1, \ldots, \mathbf{S}_k)} \tag{3.73}$$

$$= \sum_{i=1}^{k} n_i \log \frac{|\widehat{\boldsymbol{\Sigma}}_i|}{|\mathbf{S}_i|} \tag{3.74}$$

which has a chi-square distribution with $(k-1)p(p-1)/2$ degrees of freedom.

3.8 Results for the Flury Chi-squared Test

The results of the Flury chi-squared test for the comparison of depressed females to depressed males are shown in Table 3.2. The first column shows the data being compared, personality or symptoms at the particular time point. The following columns present the chi-square test statistic for the null hypothesis of common principal components versus the alternate hypothesis that the groups are unrelated, with the degress of freedom (df) and the associated p-value. The table shows quite clearly that there are common components for the baseline symptoms and no common components for the symptoms after treatment. The results for personality are not as clear. At baseline the p-value is just below the 10% significance level and just above it after treatment. With the result being so close to the significance level personality will be kept separate for males and females. Possible gender differences will be investigated further in Chapter 4 using the multigroup scenario which will allow for a thorough investigation of any differences or similarities at a number of levels.

Thus the analyses will be done separately on males and females for personality at both time points and the symptoms after treatment. The baseline symptoms will be analysed with the depressed males and females combined to give more power from the increase in sample size.

Method	DFem T0	DFem T6	DMale T0	DMale T6
PVAF	5	5	5	5
Armor's Theta	1	1	1	1
Velicer's MAP	1	1	1	1
AutoScree	4,3	1,3	2	3,2

Table 3.3: Number of components to retain for personality. Key: D = Depressed, N = Normal, Fem = Female, T0 = baseline, T6 = six months

Method	DFem T0 & DMale T0	DFem T6	DMale T6
PVAF	6	4	4
Armor's Theta	1	1	1
Velicer's MAP	1	2	1
AutoScree	4,6,2	5,3,4	3,4,5,6

Table 3.4: Number of components to retain for symptoms. Key: D = Depressed, N = Normal, Fem = Female, T0 = baseline, T6 = six months

3.9 The Number of Components Retained

Using the program PSPWIN (www.pbarrett.net) the number of factors to rotate were determined for the exploratory depressed patient datasets using Armor's Theta, Velicer's MAP, and AutoScree. SAS (SAS(R) Proprietary Software Release (8.1)) was used to calculate the percentage variance accounted for (PVAF). Particular attention is paid to the more reliable methods (MAP, AutoScree and Armor's Theta (Zwick and Velicer, 1986)). Table 3.3 shows the corresponding results for personality. The different groups are shown in the first row. The recommended number to rotate by the different methods read down each column. Table 3.4 presents the results for the symptom data formatted as above. AutoScree produces up to 4 results and these are presented in order of importance.

Component models were also developed that included both the symptom and personality data in one model to further investigate their interaction. Results from the number

Method	DFem T0	DFem T6	DMale T0	DMale T6
PVAF	7	7	7	5
Armor's Theta	2	1	1	2
Velicer's MAP	2	1	1	2
AutoScree	4, 10, 2, 3	5, 8, 4, 6	7, 3, 5, 6	3, 9, 8, 12

Table 3.5: Number of components to retain for symptoms and personality combined. Key: D = Depressed, Fem = Female, T0 = baseline, T6 = six months

of components to retain are shown in Table 3.5.

Clearly all the methods show, at times, quite different results. Armor's Theta and Velicer's MAP tend to give the smallest number. Generally Bartlett's test (not shown here) and the percentage variance accounted for (PVAF) gave the maximum number to rotate. This agrees with the findings of Zwick and Velicer (1986). The percentage variance accounted for test was retained as it is important in the sense that we want to retain as much of the information as possible. To find the best model from these, for each group, structural equation modelling will be used in Chapter 4.

3.10 Component Model Results

PC and IC analyses were conducted on the covariance structure of the exploratory datasets. However the small eigenvalues resulting from this made factor analysis within SAS's (SAS(R) Proprietary Software Release (8.1)) preset conditions difficult, so the factor analysis was conducted on the correlation matrix. At times for the larger number of components SAS could not be forced to give a solution. The eigenvalues for the correlation matrix are higher with more of them greater than one. In the next chapter confirmatory factor analysis will be conducted on the models that are generated in this chapter. The confirmatory factor analysis uses a model where each variable only loads on one factor (Chapter 4). Thus the models developed in this chapter are interpreted with each variable only allowed to load on one factor. The highest significant loading is presented in bold. In some cases this may mean that a particular component has a significant loading from a variable that is ignored as this particular variable loads higher on a different factor.

All the component models are presented in Appendix A. The best models found from the confirmatory analysis conducted in the next chapter are presented here. With each model, the corresponding models for the same number of components but from the two other component methods (i.e. PCA, ICA or FA) will also be presented for comparative purposes.

The Personality Models

Table 3.6 presents the one component solutions for the depressed females personality at baseline. The top four loadings are highlighted, as a minimum of four variables will be needed for identification of the structural model with only one component (see Section 4.1). The principal and independent component methods give the same results with a model of novelty seeking and harm avoidance versus persistence and self directedness. The factor model drops novelty seeking and has cooperativeness instead.

Table 3.7 presents the one component models for the depressed female's personality af-

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	**-0.396**	**0.704**	-0.093
Harm Avoidance	**-0.449**	**0.909**	**-0.550**
Reward Dependence	0.082	-0.152	0.021
Persistence	**0.929**	**-2.790**	**0.213**
Self Directedness	**0.530**	**-1.154**	**1.000**
Cooperativeness	0.308	-0.428	**0.439**
Self Transcendence	0.169	-0.293	0.004

Table 3.6: Depressed females TCI at baseline, one component solution.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	-0.044	-0.083	0.082
Harm Avoidance	**-0.597**	**-1.352**	**0.578**
Reward Dependence	0.035	0.068	0.058
Persistence	**0.866**	**2.817**	**-0.159**
Self Directedness	**0.581**	**1.450**	**-1.000**
Cooperativeness	**0.253**	0.351	**-0.435**
Self Transcendence	0.196	**0.405**	-0.003

Table 3.7: Depressed females TCI at six months, one component solution.

ter treatment. This time the methods of principal component analysis and factor analysis give the same results, harm avoidance is contrasted against persistence, self directedness and cooperativeness, this is the same model that factor analysis found at baseline. The independent component solution models harm avoidance versus persistence, self directedness and self transcendence.

At baseline the confirmatory factor analysis did not find a satisfactory model for the male's personality. A one component model was found to be best after treatment. The one component models tested are presented in Table 3.8. All three methods lead to the same model, harm avoidance contrasted against persistence, self directedness and cooperativeness. This is the same as the FA model for the baseline females and the FA and PC models for the post treatment females. Of note is that in all the models harm avoidance is contrasted against persistence and self directedness and the fourth variable changes between the different models. The three variables harm avoidance, persistence and self directedness are important for describing the personality of depressed patients both pre and post treatment.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	0.105	0.146	0.001
Harm Avoidance	**-0.895**	**-2.067**	**-0.674**
Reward Dependence	0.314	0.350	0.245
Persistence	**0.609**	**1.265**	**0.346**
Self Directedness	**0.852**	**1.435**	**0.995**
Cooperativeness	**0.574**	**0.758**	**0.638**
Self Transcendence	-0.061	-0.108	-0.015

Table 3.8: Depressed males TCI at six months, one component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
Somatisation	**0.890**	0.164	0.032	0.147	0.132	0.082
Obsessive Compulsive	0.261	0.148	0.235	0.126	**0.910**	0.099
Interpersonal Sensitivity	0.036	**0.722**	0.435	0.408	0.142	0.099
Depression	0.279	0.235	**0.847**	0.130	0.246	0.090
Anxiety	**0.764**	0.073	0.417	0.197	0.198	0.074
Anger Hostility	0.111	0.206	0.082	0.088	0.087	**0.960**
Phobic Anxiety	0.292	0.238	0.138	**0.890**	0.124	0.101
Paranoid Ideation	0.243	**0.902**	0.074	0.101	0.093	0.220
Psychotocism	**0.538**	0.433	0.334	0.181	0.225	0.133
SCL Symptoms	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
Somatisation	-0.333	0.284	-0.114	**0.875**	-0.136	0.548
Obsessive Compulsive	0.551	-0.004	-0.274	0.440	**1.332**	-0.644
Interpersonal Sensitivity	-0.294	0.007	-0.278	**-0.713**	-0.043	-0.324
Depression	-0.008	-0.363	0.209	**-1.152**	0.177	0.836
Anxiety	-0.119	0.184	0.119	0.055	-0.037	**0.842**
Anger Hostility	**1.129**	-0.376	-0.350	0.001	-0.566	0.262
Phobic Anxiety	0.426	**1.350**	0.021	-0.176	-0.371	-0.485
Paranoid Ideation	**-0.742**	-0.435	-0.704	0.546	-0.106	-0.337
Psychotocism	-0.237	-0.042	-0.145	0.121	0.035	0.290

Table 3.9: Depressed males and females symptoms at baseline, six component solution.

SCL Symptoms	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
Somatisation	**0.735**	0.173	**-0.573**	-0.409	**0.678**	0.223
Obsessive Compulsive	**0.884**	0.348	**-0.954**	-0.525	**0.823**	0.407
Interpersonal Sensitivity	0.390	**0.809**	0.440	**0.978**	0.378	**0.787**
Depression	**0.693**	0.666	-0.215	**0.511**	**0.676**	0.647
Anxiety	**0.873**	0.368	**-0.695**	-0.357	**0.894**	0.369
Anger Hostility	**0.674**	0.343	**-0.499**	-0.191	**0.575**	0.374
Phobic Anxiety	0.349	**0.497**	0.066	0.260	0.306	**0.507**
Paranoid Ideation	0.132	**0.893**	0.813	**1.304**	0.208	**0.759**
Psychotocism	0.461	**0.716**	0.119	0.379	0.462	**0.733**

Table 3.10: Depressed females SCL at six months, two component solution.

The Symptom Models

Table 3.9 presents the PC and IC models for six components. There were too many factors for a factor model to be calculated. The PC and IC solutions are very different. The PC solution has two mixture constructs and four constructs with one variable. The IC model has a redundant factor, a factor on which no variable has its highest loading. The IC model also has two mixture components and then three single indicator components. Interestingly, and this applies across all the symptom models not just the one presented above, the PC solutions for symptoms only have positive loadings but the IC solutions have contrasts between the solutions as demonstrated above. For example the first IC is a contrast of anger hostility against paranoid ideation.

The two component models for the depressed female's symptoms after treatment are presented in Table 3.10. The principal component and factor methods lead to the same model with the first component essentially a weighted average of somatisation, obsessive

compulsive, depression, anxiety and anger hostility. The second component is a weighted average of interpersonal sensitivity, phobic anxiety, paranoid ideation and psychotocism. The first IC is similar to the first PC and FA component but does not include depression. The second component measures a weighted average of interpersonal sensitivity, depression and paranoid ideation. The males did not have a reasonable model, under CFA testing, for their post treatment symptoms.

The Combined Personality and Symptom Models

Using confirmatory factor analysis two models were found to be suitable for describing the covariance structure of personality and symptoms combined for the depressed females at baseline. Table 3.11 presents the seven component solutions and Table 3.12 the ten component solutions. In both cases all three methods lead to different models and in some cases there are redundant factors. Table 3.13 presents the baseline six component models for the depressed males personality and symptoms. All three methods lead to different solutions and the factor analysis model interestingly has a first factor that is an average of six of the symptoms. With all these combined TCI and SCL models personality does not feature strongly. There are constructs with a combination of personality and symptom variables, but these only happen once or twice in a model. Generally the symptom weights either dominate entirely with few of the personality traits having significant loadings; or the personality traits themselves load on separate factors.

Graphical Comparison of the PC, IC and FA models

The simplest model is the one component solution for personality at baseline. This model has one component from the original seven traits. Graphically, as it is impossible to present all seven variables on the one graph, the variables have been grouped. The first graph shows novelty seeking versus harm avoidance (Figure 3.2(a)), for the depressed females, with the directions of the component from the three methods plotted. The IC component is in the negative direction compared to the PC and FA solution. A scaling ambiguity is found with the IC solutions (Roberts and Everson, 2001) so the component could be multiplied by negative one without affecting the model. The directions of the PC and IC solution are practically the same, ignoring the negative direction. The FA direction is quite different. In the second graph (Figure 3.2(b)) all three directions are very similar with the PC and FA directions almost exactly the same. However, for the third graph (Figure 3.2(c)) the IC and FA directions (ignoring the negative) are very similar and the PC direction is somewhat different.

Overall interpretation is difficult as there are seven variables to consider. However the three graphs do show the different directions taken by the different methods. No one

Variables	PC1	PC2	PC3	PC4	PC5	PC6	PC7
NS TCI	0.078	0.065	0.317	-0.088	0.034	0.126	-0.021
HA TCI	0.006	**0.486**	-0.020	0.027	0.145	-0.064	-0.133
RD TCI	0.064	0.080	0.007	0.086	-0.020	-0.032	-0.244
P TCI	0.120	**-0.445**	-0.133	0.067	0.002	-0.138	0.065
S TCI	-0.103	**-0.559**	-0.206	-0.129	0.035	-0.035	-0.130
C TCI	0.074	-0.118	-0.377	-0.067	-0.013	-0.033	-0.272
ST TCI	0.345	-0.019	0.372	0.109	-0.015	0.127	-0.040
S SCL	**0.944**	0.006	0.102	0.000	0.198	0.113	-0.054
OC SCL	0.321	0.102	0.202	0.226	0.085	**0.883**	0.067
IS SCL	0.272	**0.826**	0.085	0.250	0.247	0.048	0.294
D SCL	0.358	0.349	0.138	**0.761**	0.131	0.293	-0.059
A SCL	**0.717**	-0.140	0.041	0.561	0.183	0.147	-0.021
AH SCL	0.127	0.173	**0.949**	0.190	0.073	-0.065	0.080
PA SCL	0.256	0.254	0.109	0.117	**0.900**	0.086	0.146
PI SCL	0.326	0.303	0.219	0.314	0.142	-0.089	**0.788**
P SCL	**0.666**	0.358	0.157	0.294	0.038	0.159	0.112
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7
NS TCI	0.059	-0.116	0.045	0.072	-0.013	0.013	-0.100
HA TCI	0.005	-0.080	0.217	-0.208	0.094	0.119	-0.144
RD TCI	0.050	0.048	0.134	-0.110	0.040	0.107	-0.047
P TCI	-0.047	0.354	-0.204	0.158	-0.066	-0.172	0.191
S TCI	0.042	0.165	-0.092	0.130	0.050	-0.153	0.133
C TCI	-0.037	0.040	0.111	-0.038	0.044	0.039	0.006
ST TCI	0.055	-0.009	0.047	0.076	-0.064	0.046	-0.068
S SCL	-0.466	-0.097	**1.021**	0.734	-0.344	0.138	-0.732
OC SCL	0.351	**-1.250**	-0.153	0.647	-0.231	-0.269	0.696
IS SCL	-0.470	**-0.678**	0.479	-0.810	-0.058	0.287	-0.482
D SCL	0.516	0.683	0.066	**-1.029**	-0.004	0.351	0.743
A SCL	0.089	**1.049**	0.046	-0.015	-0.242	-0.091	0.539
AH SCL	**1.021**	0.117	-0.194	0.085	-0.279	0.190	-0.803
PA SCL	0.441	0.208	0.167	-0.069	0.773	**-1.315**	-0.049
PI SCL	-0.687	0.178	**-1.221**	0.393	-0.776	-0.642	0.292
P SCL	-0.296	-0.132	0.425	0.000	-0.362	0.354	-0.246
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7
NS TCI	0.131	-0.013	-0.245	-0.106	**0.764**	0.248	-0.035
HA TCI	0.019	0.136	**0.985**	0.058	-0.045	-0.079	0.012
RD TCI	0.029	0.018	0.018	**0.495**	0.027	0.091	0.034
P TCI	0.083	-0.136	-0.232	-0.010	**-0.625**	0.054	-0.074
S TCI	-0.066	-0.368	**-0.543**	0.274	-0.175	-0.173	-0.118
C TCI	0.029	-0.172	-0.085	**0.940**	-0.156	-0.221	-0.077
ST TCI	0.319	0.073	-0.179	0.238	0.016	**0.533**	0.050
S SCL	**0.847**	0.181	0.001	0.046	-0.063	0.118	-0.089
OC SCL	**0.508**	0.146	0.001	-0.029	0.141	0.129	0.361
IS SCL	0.197	**0.902**	0.275	0.048	0.157	0.061	0.204
D SCL	0.520	0.330	0.119	0.010	0.016	0.165	**0.761**
A SCL	**0.805**	0.108	-0.010	0.069	-0.070	0.110	0.232
AH SCL	0.139	0.231	0.120	-0.166	0.143	**0.717**	0.107
PA SCL	0.398	**0.477**	0.161	-0.013	0.085	0.053	0.035
PI SCL	0.249	**0.692**	0.014	-0.168	-0.034	0.246	0.069
P SCL	**0.615**	0.383	0.062	-0.015	0.141	0.194	0.199

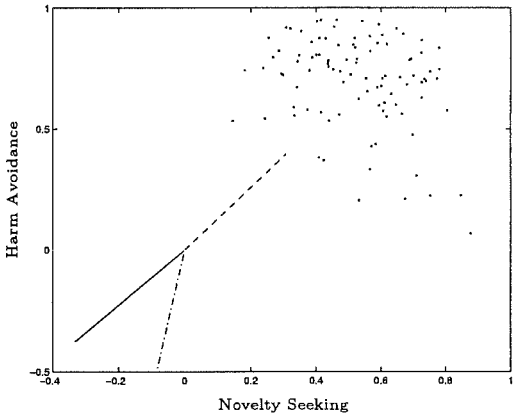
Table 3.11: Depressed females TCI and SCL at baseline, seven component solution.

Variables	PC1	PC2	PC3	PC4	PC5	PC6	PC7	PC8	PC9	PC10
NS TCI	-0.036	0.065	0.197	**-0.555**	0.069	-0.028	0.051	-0.010	0.100	-0.111
HA TCI	**0.503**	-0.008	-0.028	-0.071	-0.053	0.030	0.119	-0.071	-0.044	0.196
RD TCI	0.134	0.021	0.030	0.103	0.015	0.016	-0.033	-0.066	-0.017	**0.472**
P TCI	-0.295	0.110	0.063	**0.923**	-0.018	-0.047	0.014	-0.002	0.054	0.090
S TCI	**-0.546**	0.018	-0.205	0.108	-0.083	-0.062	0.021	-0.125	-0.182	0.007
C TCI	-0.077	0.089	-0.355	0.182	-0.046	-0.043	-0.031	-0.163	-0.013	0.321
ST TCI	-0.001	0.265	**0.404**	0.119	0.195	0.074	0.009	-0.017	0.224	0.071
S SCL	0.060	**0.937**	0.098	0.022	0.168	0.131	0.175	0.075	0.158	0.041
OC SCL	0.083	0.209	0.131	-0.119	**0.915**	0.226	0.103	0.050	0.125	0.015
IS SCL	**0.863**	0.132	0.097	-0.051	0.134	0.175	0.238	0.323	0.108	-0.014
D SCL	0.337	0.208	0.189	0.013	0.321	**0.803**	0.128	0.095	0.186	-0.011
A SCL	-0.097	**0.511**	0.052	-0.055	0.223	0.485	0.198	0.376	0.263	0.433
AH SCL	0.175	0.077	**0.946**	-0.180	0.037	0.089	0.087	0.129	0.029	0.035
PA SCL	0.277	0.204	0.095	-0.050	0.107	0.113	**0.906**	0.124	0.074	-0.062
PI SCL	0.357	0.155	0.265	0.047	0.049	0.130	0.172	**0.780**	0.145	-0.310
P SCL	0.289	0.360	0.165	-0.138	0.192	0.250	0.118	0.171	**0.770**	-0.064
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8	IC 9	IC 10
NS TCI	-0.053	0.229	0.120	0.129	-0.067	**-0.830**	0.158	0.161	0.527	0.501
HA TCI	0.123	-0.106	-0.291	-0.168	-0.148	0.326	0.139	0.080	0.141	0.103
RD TCI	0.257	-0.371	-0.297	-0.237	-0.248	**0.693**	0.364	-0.116	-0.176	-0.143
P TCI	0.081	-0.789	-0.173	-0.064	0.035	2.116	-0.669	-0.475	**-2.126**	-1.484
S TCI	0.009	0.053	0.135	0.127	0.319	-0.171	-0.002	0.017	0.063	-0.113
C TCI	0.058	-0.219	-0.179	-0.106	-0.084	0.340	0.126	-0.061	-0.102	-0.107
ST TCI	0.049	-0.164	-0.086	-0.035	-0.143	0.274	-0.141	-0.038	-0.465	-0.166
S SCL	-0.601	0.618	-0.918	-0.549	0.924	-0.778	-0.358	**1.221**	-0.264	-0.083
OC SCL	0.327	0.339	-0.088	0.139	-0.904	-0.266	0.226	0.045	0.058	**-1.322**
IS SCL	-0.108	0.218	-0.930	-0.660	-0.634	**1.266**	0.334	0.212	0.366	0.078
D SCL	0.393	0.537	0.262	-0.104	1.305	-0.343	**-1.724**	-0.608	0.471	0.275
A SCL	0.599	-0.847	-0.213	-0.345	0.112	0.334	**1.950**	-1.025	0.423	0.216
AH SCL	**1.012**	0.217	0.113	-0.129	-0.120	0.183	0.237	0.432	-0.646	0.423
PA SCL	0.197	-0.325	-0.134	**1.608**	0.258	0.073	-0.088	0.093	0.026	0.059
PI SCL	-0.830	0.986	**1.009**	0.233	0.509	-0.407	0.493	-0.395	-0.207	-0.396
P SCL	-0.663	-0.946	-0.088	0.376	**-1.541**	-0.473	-0.673	-0.859	-1.243	0.882
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8	FA 9	FA 10
NS TCI	0.121	0.025	**-0.444**	-0.387	0.022	-0.316	0.140	-0.094	-0.087	-0.064
HA TCI	0.028	0.182	-0.083	**0.938**	0.030	-0.012	-0.087	-0.263	-0.024	0.014
RD TCI	0.046	0.012	0.007	0.012	**0.992**	0.091	0.051	-0.007	-0.038	0.021
P TCI	0.047	-0.098	**0.979**	-0.111	0.022	0.049	0.077	0.054	-0.032	-0.064
S TCI	-0.096	-0.160	0.099	-0.304	0.008	0.281	0.011	**0.843**	-0.119	-0.095
C TCI	0.024	-0.062	0.055	0.077	0.368	**0.658**	0.152	0.292	-0.085	-0.049
ST TCI	0.272	0.028	0.045	-0.121	0.069	-0.083	**0.944**	0.019	0.029	0.072
S SCL	**0.839**	0.176	0.081	0.002	0.006	-0.022	0.139	0.003	0.017	-0.090
OC SCL	**0.520**	0.114	-0.122	-0.049	-0.008	-0.113	0.144	-0.089	-0.032	0.348
IS SCL	0.230	**0.788**	-0.160	0.165	0.054	-0.067	0.053	-0.300	0.300	0.237
D SCL	0.551	0.242	-0.023	0.078	0.036	-0.132	0.086	-0.117	0.120	**0.760**
A SCL	**0.833**	0.027	0.046	0.007	0.102	-0.020	0.057	0.028	0.139	0.209
AH SCL	0.173	0.149	-0.118	0.043	0.072	**-0.620**	0.302	-0.073	0.185	0.111
PA SCL	0.406	**0.520**	-0.039	0.131	-0.030	-0.161	0.009	0.029	0.119	0.037
PI SCL	0.271	0.403	0.016	-0.011	-0.086	-0.256	0.048	-0.146	**0.801**	0.074
P SCL	**0.638**	0.251	-0.130	-0.017	-0.040	-0.082	0.167	-0.242	0.211	0.193

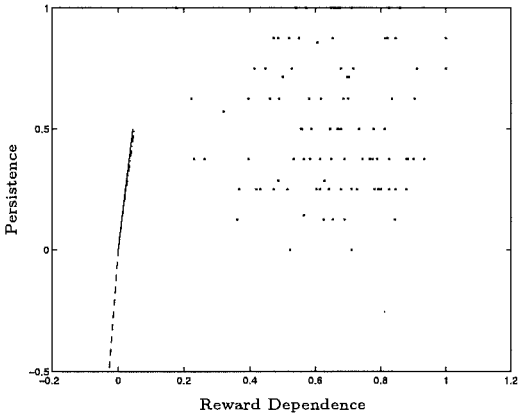
Table 3.12: Depressed females TCI and SCL at baseline, ten component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
NS TCI	-0.127	0.189	0.084	-0.013	-0.113	-0.036
HA TCI	0.292	0.017	0.140	0.192	0.145	0.119
RD TCI	0.054	0.017	0.034	0.007	0.177	-0.165
P TCI	0.005	-0.010	0.189	0.067	0.204	-0.009
S TCI	-0.389	-0.016	-0.200	-0.108	-0.352	-0.162
C TCI	-0.080	-0.174	**-0.437**	-0.115	-0.121	-0.200
ST TCI	0.027	0.271	0.001	0.057	0.032	0.143
S SCL	0.151	**0.912**	0.137	0.108	0.110	0.031
OC SCL	0.337	0.208	0.116	0.142	**0.888**	0.130
IS SCL	0.547	0.061	0.214	0.430	0.191	**0.581**
D SCL	**0.946**	0.133	0.001	0.115	0.148	0.046
A SCL	0.511	**0.585**	0.108	0.384	0.256	-0.174
AH SCL	0.108	0.112	**0.982**	0.043	0.089	0.024
PA SCL	0.277	0.303	0.156	**0.851**	0.152	0.105
PI SCL	0.365	0.354	0.331	0.084	0.075	**0.763**
P SCL	0.486	**0.500**	0.176	0.330	0.181	0.226
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
NS TCI	0.013	0.010	-0.056	-0.059	0.030	0.067
HA TCI	-0.026	-0.067	0.052	0.080	0.009	-0.046
RD TCI	-0.052	0.011	0.035	0.034	-0.031	0.023
P TCI	-0.166	0.007	0.068	-0.018	-0.006	-0.035
S TCI	0.061	-0.033	-0.109	-0.096	0.010	0.047
C TCI	0.063	-0.009	0.002	0.029	-0.061	-0.005
ST TCI	0.044	0.051	-0.013	-0.111	0.006	0.041
S SCL	0.324	0.482	-0.213	-0.635	0.044	**0.927**
OC SCL	-0.850	0.751	**1.042**	-0.336	-0.543	-0.229
IS SCL	0.150	-0.425	0.289	0.004	0.213	**-0.682**
D SCL	0.687	-0.109	0.251	**1.458**	-0.042	0.300
A SCL	0.106	-0.224	0.046	0.184	-0.191	**0.741**
AH SCL	**-0.881**	-0.118	-0.188	0.645	0.803	0.321
PA SCL	-0.343	**-1.257**	-0.057	-0.681	-0.109	-0.184
PI SCL	0.576	0.608	0.020	-0.504	**0.621**	-0.210
P SCL	0.234	-0.038	0.026	-0.075	0.055	0.282
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6
NS TCI	0.096	**0.612**	0.021	-0.182	-0.346	0.102
HA TCI	0.208	**-0.667**	0.090	-0.197	0.064	-0.075
RD TCI	0.073	0.071	-0.027	0.059	0.129	**0.646**
P TCI	0.016	-0.194	-0.008	-0.088	**0.405**	0.187
S TCI	-0.282	**0.632**	-0.158	0.356	0.029	-0.042
C TCI	-0.178	0.324	-0.048	**0.753**	0.033	0.426
ST TCI	0.148	-0.013	0.089	0.021	**0.631**	0.028
S SCL	**0.665**	0.170	0.060	-0.242	0.216	-0.070
OC SCL	**0.545**	-0.274	0.196	-0.211	0.035	0.246
IS SCL	0.513	-0.360	**0.644**	-0.119	-0.076	-0.046
D SCL	**0.610**	-0.244	0.340	0.050	-0.029	0.144
A SCL	**0.863**	-0.101	0.003	-0.093	0.162	0.122
AH SCL	0.192	-0.039	0.190	**-0.582**	0.056	0.075
PA SCL	**0.707**	-0.108	0.201	-0.095	0.018	0.015
PI SCL	0.349	0.011	**0.837**	-0.335	0.248	-0.056
P SCL	**0.719**	-0.158	0.274	-0.281	0.022	-0.172

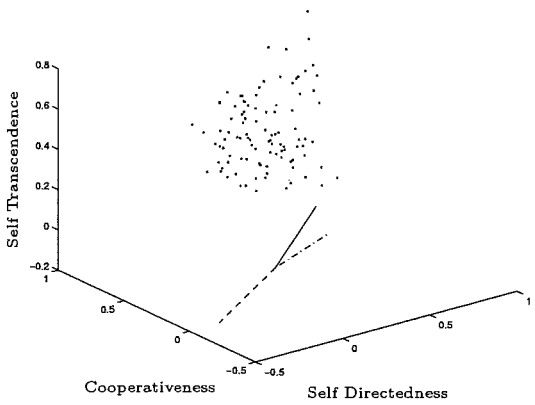
Table 3.13: Depressed males TCI and SCL at baseline, six component solution.



(a) Novelty Seeking versus Harm Avoidance



(b) Reward Dependence versus Persistence



(c) The Character Traits

Figure 3.2: Plots presenting the direction of the components compared to the original data. Key: — represents the PC component, – – represents the IC component, – · represents the FA component.

method appears to stand out, ignoring the negative difference between the IC solution and the PC and FA solutions. Each method however, is picking out different properties of the data to align with, hence the solutions are different.

3.11 Summary

This chapter used three methods to develop models that describe the underlying personality and symptom structure. The three methods were principal component analysis, factor analysis and independent component analysis. Whilst many studies on personality and symptoms of depression have used principal component (PCA) or factor analytic (FA) methods to analyse this type of data, none, as far as the author is aware, have used the recent advance of independent component analysis (ICA).

The Flury test was used initially to determine if there were significant differences between males and females on personality and symptoms. The results showed that, in all but one case, there are significant differences, or very nearly significant, across gender. Males and females need to be analysed separately if PCA, FA or ICA are used. The exception is for the males and females baseline symptoms wherein gender can be combined. The test found that there was a common underlying component structure for males and females. This lead to males and females being combined for the baseline symptoms to add more power by the increase in sample size.

Not only were three different methods used to investigate the component or factor structure, different methods were used to calculate the number of components to retain. These methods will be compared in Chapter 4 using a confirmatory factor analysis approach with the confirmatory dataset introduced in Chapter 2.

The models presented in this chapter show that the three component methods, PCA, FA and ICA, lead to, at times, quite different solutions. For the personality data, the three methods produced more similar results across methods. The symptom data however, showed some striking differences across the analytic methods. Principal component analysis and factor analysis both produced only positive loadings for the symptom variables on the underlying factors. The symptoms are expected to be positively correlated, as a person suffering under psychological distress will score highly on all the symptoms, compared to the normal population. The symptoms are considered additive and often a total symptom score is calculated. The strictly positive loadings reflect this nature of the symptoms. However, ICA produced some factors that were contrasts between the symptom variables.

The independent components are harder to interpret with variables often loading highly on more than one factor. The loadings are not bounded between zero and one, and by the nature of the ICA process, can be multiplied by a constant without affecting the

model. This makes it harder to immediately see underlying patterns. The component's loadings are at times very small compared to the PCA and FA models and at other times much larger. This is deceptive when the loadings are viewed in the same table as the PCA and FA solutions. With the confirmatory factor analysis technique utilised in Chapter 4, variables were only allowed to load on to one underlying factor. This is an area that can be extended in future work by allowing the variables to cross load onto the factors. This makes the factors harder to interpret and the subsequent modelling more complicated requiring a larger sample size.

All the models developed are presented Appendix A. To investigate which model, out of all the possible models (across methods and number of components retained), is the best model for describing the covariance structure, confirmatory factor analysis is needed. Chapter 4 compares the models developed in this chapter finding the most reasonable models for each group. These models are then investigated further in a multigroup and a longitudinal context. This leads to the analysis conducted in chapter 5 that investigates the relationship between personality and symptoms.

This study is different to most of the studies in the literature, which investigate the factor structure of the questionnaires rather than the traits. This study had a relatively small sample size making analysis of the original questionnaire data unadvisable. The trait structure, which has been well documented in the literature, was used directly to investigate the underlying structure. Rather than looking at the questions that make up the traits, the interactions between the traits are being investigated. It is easier to model the traits, as a continuous model can then be used, rather than modelling the discrete questionnaire outcomes.

Chapter 4

Confirmatory Modelling of the latent personality and symptom structures

Structural equation modelling (SEM) (Jöreskog, 1970; Duncan, 1975) investigates how well structures, or models, developed either theoretically or by factor analysis, fit the population of interest. A number of models were developed in Chapter 3 using principal component analysis (PCA), independent component analysis (ICA) and factor analysis (FA), however, the methods could not show which of the models was the best for each group (grouped by gender and time point). Structural equation modelling will allow us to validate these models on a second data set and confirm which of the possible models best describes the personality and symptom data of the depressed patients.

Structural equation modelling has applications in a wide range of fields including biology (Torres et al., 2002; Svensson et al., 2001; Shipley, 1999; Mitchell, 1994), sports medicine (Motl et al., 2003; Maia et al., 2001) and health (Bennett et al., 2002; McManus et al., 2002; Sharkey, 2002; Oxford et al., 2001). Coovert et al. (1990) conducted a survey that showed SEM remained largely unknown in the personality/social research area up until 1990. Jackson et al. (2000) used structural equation modelling to investigate the structure of the Eysenck Personality Profiler. Studies, such as those by Heaven (1996) and Ormel et al. (1989), have used personality as a variable in structural equation modelling. Structural equation modelling has been used more frequently in the depression area to model relationships between depression and various psychological, physical and genetic factors (Lonigan et al., 2003; McCaffery et al., 2003; Fu et al., 2002).

SEM starts with the formulation of a structural model. This model can be developed from a theoretical model or from traditional factor analysis or components analysis as presented here. The model may contain latent (underlying) factors and manifest (observed) variables each with their own errors and uncertainties. The structural model will show relationships via covariances, correlations or regression coefficients between latent

variables.

Structural models are converted to mathematical language as a series of linear equations, which describe the theoretical covariance structure. Comparison and minimisation between the theoretical and sample covariance structure provides the model parameter solutions. A number of different indicators have been developed such as the normed fit index (Bentler and Bonett, 1980), the comparative fit index (Bentler, 1990) and the Akaike information criterion (Akaike, 1973), to describe the model fit.

This chapter initially uses a specific formulation of the SEM model, namely that of confirmatory factor analysis (CFA). CFA investigates how well the underlying latent structures developed in Chapter 3 and Appendix A generalise to a second dataset. A good fit indicates that the covariance matrix implied by the structural model matches that seen in the data. This test will indicate which of the models, from the varying number of components retained and different component methods used, is the best model for the data. Once the best models have been found the chapter will investigate gender differences between the structures using multigroup analysis and extend the models to investigate changes across time in a longitudinal framework. The final section of this chapter investigates the interpretability of the factors that have been developed, using both discriminant analysis and logistic regression.

4.1 The Theory Behind Structural Equation Modelling

Structural equation modelling (SEM) (Jöreskog, 1970; Duncan, 1975) is used in this chapter to test the validity of a number of personality and symptom models. The models describe the relationships between the manifest and latent variables. The manifest variables are the observed variables. In this study these are the personality traits and symptom scores. The latent variables are the hypothetical underlying factors or components, which in this case are the components calculated from PCA, ICA and FA. SEM investigates covariances (Everitt, 1984) and unlike multiple regression and other multivariate techniques (Cliff, 1987), SEM is not limited to investigating manifest variables.

The following theory develops the steps needed to get from the structural model to the predicted model covariance matrix, to the estimation of the model parameters. After the model parameters have been estimated the plausibility of the model is investigated using model fit indices. This section follows the theory of Everitt (1984) and Bollen (1989). They both use the common LISREL (Jöreskog, 1973; Keesling, 1972; Wiley, 1973) notation.

Structural Equation Modelling tests the hypothesis that the population covariance matrix ($\boldsymbol{\Sigma}$) is equal to the covariance matrix predicted from the model ($\boldsymbol{\Sigma}(\boldsymbol{\theta})$), where $\boldsymbol{\theta}$ is the vector of model parameters. The model consists of two parts, the measurement model

and the structural model. The measurement model describes the relationships between the latent and manifest variables and the structural model describes the relationship between the latent variables. These models can be represented graphically in path diagrams (see Figure 4.1).

The structural part of the model consists of two types of latent variables, independent ($\boldsymbol{\xi}$) and dependent ($\boldsymbol{\eta}$), and are related by the following linear equation Bollen (1989)

$$\boldsymbol{\eta} = B\boldsymbol{\eta} + \Gamma\boldsymbol{\xi} + \boldsymbol{\zeta}, \tag{4.1}$$

where $\mathbf{B}$ are the regression elements of the direct effects of the dependent latent variables ($\boldsymbol{\eta}$) on other dependent latent variables ($\boldsymbol{\eta}$); the matrix $\boldsymbol{\Gamma}$ contains the regression elements of the direct effects of the independent variables ($\boldsymbol{\xi}$) on the dependent latent variables ($\boldsymbol{\eta}$); the vector $\boldsymbol{\zeta}$ contains the errors and random disturbances which are uncorrelated with the independent latent variables.

The measurement model is used to relate the manifest variables to the latent variables. Equations 4.2 and 4.3 represent the relationship between the manifest variables ($\boldsymbol{x}$ and $\boldsymbol{y}$) and the latent variables ($\boldsymbol{\xi}$ and $\boldsymbol{\eta}$)

$$\boldsymbol{y} = \Lambda_{\boldsymbol{y}}\boldsymbol{\eta} + \boldsymbol{\epsilon} \tag{4.2}$$

$$\boldsymbol{x} = \Lambda_{\boldsymbol{x}}\boldsymbol{\xi} + \boldsymbol{\delta} \tag{4.3}$$

where $\boldsymbol{\Lambda_y}$ and $\boldsymbol{\Lambda_x}$ are matrices of regression weights showing the effects of the independent latent variables ($\boldsymbol{\eta}$) on their associated manifest variables ($\mathbf{y}$) and similarly the dependent latent variables ($\boldsymbol{\xi}$) on their manifest variables ($\mathbf{x}$). The residual terms $\boldsymbol{\epsilon}$ and $\boldsymbol{\delta}$ contain the measurement error in the manifest variables and are uncorrelated with the latent variables and their associated error terms.

The predicted model covariance matrix of the $\mathbf{x}$ variables $\boldsymbol{\Sigma_{xx}}$ is given by (Everitt, 1984)

$$\begin{aligned}
\boldsymbol{\Sigma_{xx}} &= E(\boldsymbol{xx'}) \\
&= E([\Lambda_{\boldsymbol{x}}\boldsymbol{\xi} + \boldsymbol{\delta}][\Lambda_{\boldsymbol{x}}\boldsymbol{\xi} + \boldsymbol{\delta}]') \\
&= E(\Lambda_{\boldsymbol{x}}\boldsymbol{\xi}\boldsymbol{\xi}'\Lambda_{\boldsymbol{x}}' + \Lambda_{\boldsymbol{x}}\boldsymbol{\xi}\boldsymbol{\delta}' + \boldsymbol{\delta}\boldsymbol{\xi}'\Lambda_{\boldsymbol{x}}' + \boldsymbol{\delta}\boldsymbol{\delta}') \\
&= E(\Lambda_{\boldsymbol{x}}\boldsymbol{\xi}\boldsymbol{\xi}'\Lambda_{\boldsymbol{x}}') + E(\Lambda_{\boldsymbol{x}}\boldsymbol{\xi}\boldsymbol{\delta}') + E(\boldsymbol{\delta}\boldsymbol{\xi}'\Lambda_{\boldsymbol{x}}') + E(\boldsymbol{\delta}\boldsymbol{\delta}').
\end{aligned}$$

Now $\boldsymbol{\delta}$ is assumed to be uncorrelated with $\boldsymbol{\xi}$ so

$$E(\Lambda_{\boldsymbol{x}}\boldsymbol{\xi}\boldsymbol{\delta}') = 0 = E(\boldsymbol{\delta}\boldsymbol{\xi}'\Lambda_{\boldsymbol{x}}') \tag{4.4}$$

giving

$$\Sigma_{xx} = E(\Lambda_x \xi \xi' \Lambda_x') + E(\delta \delta'). \tag{4.5}$$

Substituting $\Phi = E(\xi\xi')$ (the covariance matrix for the independent latent variables) and $\theta_\delta = E(\delta\delta')$ (the covariance matrix for the errors in the independent variables) gives

$$\Sigma_{xx} = \Lambda_x \Phi \Lambda_x' + \theta_\delta. \tag{4.6}$$

The same process is used to predict the covariance matrix for y and the covariances between the x's and y's (xy) from the model giving

$$\Sigma_{yy} = \Lambda_y (I - B)^{-1} (\Gamma \Phi \Gamma' + \Psi)(B')^{-1} \Lambda_y' + \theta_\epsilon \tag{4.7}$$

$$\Sigma_{xy} = \Lambda_y (I - B)^{-1} \Gamma \Phi \Lambda_x' \tag{4.8}$$

where $\Psi = E(\zeta\zeta')$ is the covariance matrix for the dependent latent errors and $\theta_\epsilon = E(\epsilon\epsilon')$ is the covariance matrix for the errors in the dependent variables. Note that $(I - B)$ is assumed to be non-singular so that an inverse exists.

Purely from the model the predicted covariance matrices for the observed variables has been derived. If we had the correct model and knew the parameters (Λ_y, Λ_x, B, Γ, Φ, Ψ, θ_ϵ, θ_δ) the predicted covariance matrix would in fact be the population covariance matrix.

The model parameters are either fixed, constrained or free. Fixed parameters are those that are fixed to a numeric value, constrained parameters are those that are set equal to some other parameter, and free parameters are those that need to be estimated. The unknown free parameters are estimated by minimising the difference between the sample covariance matrix ($\mathbf{S}$) and the predicted covariance matrix ($\Sigma(\theta)$). The techniques used for minimisation are discussed further on page 89.

Confirmatory Factor Analysis

Confirmatory Factor Analysis (CFA) (Kline, 1998) is a specific type of structural model. The variances of the latent variables are fixed to be one and these variables are allowed to covary, with no regression elements between them.

Structural Diagrams

The structural model can be represented graphically in a path diagram (see Figure 4.1). The general notation for the path diagram is for the latent variables to be in circles or ovals (⬭), the manifest variables in boxes (▭) and the error, or disturbance terms, are not

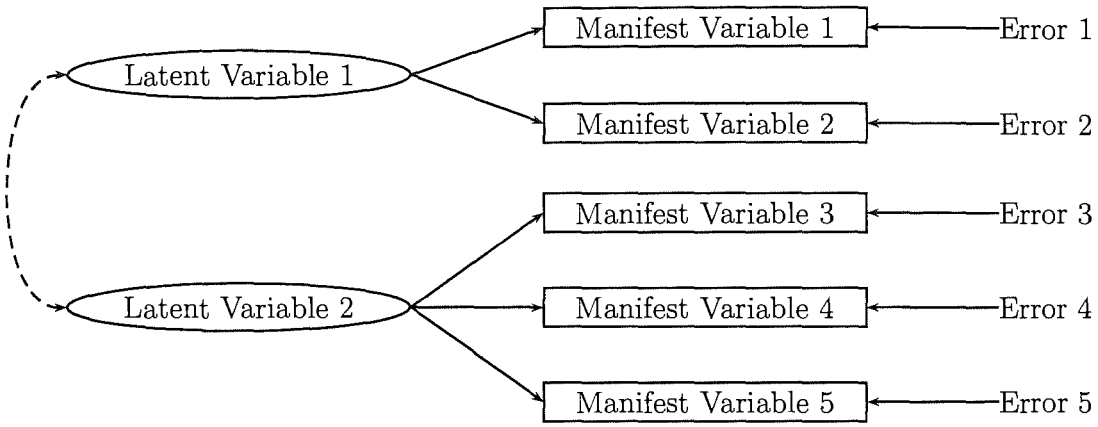


Figure 4.1: Path Diagram

enclosed. A straight arrow (→) shows a directional relationship between variables, whilst a curved double headed arrow (↜↝) represents a correlation or covariance between variables.

This path diagram forms a set of linear equations. To solve these uniquely, the model must be identified. For two parameter vectors $\boldsymbol{\theta_1}$ and $\boldsymbol{\theta_2}$ the model is identified if and only if (Everitt, 1984),

$$\text{when } \Sigma(\boldsymbol{\theta_1}) = \Sigma(\boldsymbol{\theta_2}) \tag{4.9}$$

$$\text{then } \boldsymbol{\theta_1} = \boldsymbol{\theta_2}. \tag{4.10}$$

The conditions required for this to be true are not obvious with structural equation modelling so a number of tests have been developed to check for identification. One important identification criteria is that the number of parameters must be less than or equal to the number of distinct entries in the predicted covariance matrix (Coovert et al., 1990; Bollen, 1989). The number of distinct entries (N_e) predicted, is given by

$$N_e = \frac{(p+q)(p+q+1)}{2} \tag{4.11}$$

where p and q are the number of independent and dependent measured variables respectively.

4.1.1 Estimation Methods

There are a number of different ways of finding the estimates for the model parameters. The most common methods are maximum likelihood (ML) estimation and generalised least squares (GLS) estimation. Both of these methods give estimates that are unbiased for large samples, consistent, efficient and as the sample size increases the estimator

distribution approximates a normal distribution (Bollen, 1989). These properties depend on assumptions regarding large sample size, continuous variables, multivariate normality and validity of the model being met (Bollen, 1989; West et al., 1995).

Maximum Likelihood

Maximum likelihood estimation finds parameters such that the likelihood of obtaining the sample covariance matrix ($\mathbf{S}$) from the population ($\mathbf{\Sigma}$) is maximised. This is equivalent to finding the parameter estimates that are most likely to be caused by sampling error. The formula for the maximum likelihood fitting function (Bollen, 1989) is

$$F_{ML} = \log|\Sigma(\theta)| + tr(S\Sigma^{-1}(\theta)) - \log|S| - (p+q). \tag{4.12}$$

The likelihood function is maximised when this fit function is at a minimum. Therefore F_{ML} is zero (perfect fit) when the model is a perfect predictor of the sample covariance matrix.

Generalised Least Squares

Generalised least squares estimates parameters by minimising the sum of squares difference between the observed and estimated covariance matrix. Jöreskog and Goldberger (1972) introduced the generalised least squares method to the area of exploratory factor analysis. The following formula for generalised least squares is from Bollen (1989) and West et al. (1995).

$$F_{GLS} = (\frac{1}{2})tr(([S - \Sigma(\theta)]W^{-1})^2) \tag{4.13}$$

where W^{-1} is a weight matrix. The most common choice is $W^{-1} = S^{-1}$.

In the Jöreskog and Goldberger (1972) paper the GLS method for exploratory factor analysis is shown to be scale free and, with the assumption of normality, the estimates have the same asymptotic properties as those found using maximum likelihood estimation. The paper (page 245) also shows that "the ML [Maximum Likelihood] criterion can be viewed as an approximation to the GLS criterion". These estimation methods are run iteratively until the solution converges to some desired level.

This chapter uses maximum likelihood estimation within SAS PROC CALIS (SAS(R) Proprietary Software Release (8.1)) and Mplus (Muthén and Muthén, Version 2, www.statmodel.com). Statistica's SEPATH (StatSoft, Inc.) procedure used generalised least squares to obtain initial estimates for the maximum likelihood estimation.

The maximum likelihood method for model estimation relies on the assumption of normality for the manifest variables. When the dataset violates this multivariate normality assumption, an alternative estimation method can be used. The asymptotically

distribution free estimator (ADF) method was introduced by Browne (1984). It is based on generalised least squares but uses a more complex weight matrix. This weight matrix reduces to that of the standard GLS matrix, when the data is multivariate normal. The drawback to this method is that it requires a large data set for stability of estimates (Jöreskog and Sörbom, 1992) and it requires intensive computations that become impractical for more than 20 to 25 measured variables (Bentler, 1989). Thus the ADF method can not been used for this study, even though it would be better than ML estimation as the data is not multivariate normal in some instances.

4.1.2 Evaluation of the Model: Fit Indices

A number of different measures are available to test the fit of the model to give an indication of how well the model fits the sample. These indices reflect different aspects of model fit (Kline, 1998), so a range of indices are reported. The next section discusses each of the fit indices used in this study.

The Chi-Square Statistic

One indicator used for testing model fit is the Chi-square statistic (Bollen, 1989). The maximum likelihood and generalised least squares fitting function, when multiplied by $(N-1)$, have an approximate chi-square distribution (Bollen, 1989). This distribution has degrees of freedom given by:

$$df = [(p+q)(p+q+1)]/2 - t \tag{4.14}$$

where t is the number of free parameters, p is the number of independent measured variables and q is the number of dependent measured variables.

A significant Chi-square means that the estimated covariance matrix is significantly different to the sample covariance matrix (Kline, 1998). In some situations the Chi-square turns out to be a poor indicator of fit. When the sample size is large, the power of the test is high and a model with only small differences will be rejected. Conversely, when the sample size is small, the power of the test is small thus increasing the occurrence of type II errors (Hayduk, 1987; Kline, 1998).

The Normed Fit Index

Bentler and Bonett (1980) developed two fit indices that use relative measures. They use a null model for comparison to the model of interest. Bentler and Bonett (1980) propose the null model, as the most restricted model, which means the variables are mutually independent. They also show that the null model can be obtained using

$$\Sigma = \Phi, \tag{4.15}$$

where Φ (which is the covariance matrix for the independent latent variables) is a diagonal matrix, except when parameters of little interest are fixed. The fit index introduced is called Δ (delta) or the normed fit index (NFI) (Bentler and Bonett, 1980). This fit index represents an overall fit of the model of interest (l) in relation to the null model (0)

$$\Delta_{k,l} = \frac{F_k - F_l,}{F_0} \tag{4.16}$$

where F is the minimum value that the fitting function obtains, the fitting function can be generalised least squares, maximum likelihood or unweighted least squares. Bentler and Bonett (1980) showed that the normed fit index must lie in the range (0,1). They suggested that the NFI should be greater than 0.9 for a model to be a reasonable representation of the population.

Root Mean Square Residual (RMSR or RMR)

As the name suggests this measure of fit looks at the root mean square residual between the sample and estimated covariance matrices. The difference between each element is calculated and squared. The sum is taken and then divided by the number of variances and covariances (N). This is then square rooted to give the fit index, RMSR, where

$$RMSR = \sqrt{\frac{\Sigma(S_{I,j} - \widehat{\Sigma}_{I,j})^2}{N}} \tag{4.17}$$

When the residual matrix is small (i.e. the difference between the estimated and sample covariance matrix is small) then RMSR will be small. The limiting case is zero, which would indicate perfect fit.

The standardised RMSR or RMR uses the standardised residuals (residuals divided by the residual's standard error) to calculate the root mean square residual.

Steiger-Lind RMSEA Index

RMSEA stands for root mean squared error approximation and is defined as (Steiger and Lind, 1980)

$$RMSEA = \sqrt{\max\left(\frac{F}{df} - \frac{1}{NM}, 0\right)} \tag{4.18}$$

where NM is equal to $(N-1)$ when a covariance or correlation matrix is analysed. This is a measure of approximate fit rather than exact fit, such as the chi-square measure, and is the misfit per degrees of freedom.

Jöreskog GFI

The Goodness-of-fit Index, developed by Jöreskog and Sörbom (1996), is formulated as follows;

$$GFI = 1 - \frac{(S - \hat{\sigma})'W^{-1}(S - \hat{\sigma})}{S'W^{-1}S} \tag{4.19}$$

where

$$(S - \hat{\sigma})'W^{-1}(S - \hat{\sigma}) = F_{MIN} \tag{4.20}$$

with F_{MIN} the fit function minimum after the model has been fitted and

$$S'W^{-1}S = F_{\text{before model fit}} \tag{4.21}$$

Jöreskog AGFI

To take into account the degrees of freedom the GFI is adjusted to

$$AGFI = 1 - \frac{(p+q)(p+q+1)}{2d}(1 - GFI) \tag{4.22}$$

where d is the degrees of freedom of the model. Both the AGFI and GFI are expected to be between zero and one.

Akaike Information Criterion (AIC)

Akaike (1973) introduced the information criterion and Akaike (1987) extended its use. The AIC is a measure of the badness of fit and is used when maximum likelihood estimation is used for parameter estimation.

$$AIC = (-2)\ln(\text{Maximum Likelihood}) + 2(\text{Number of Parameters}) \tag{4.23}$$

This measure of fit was introduced in an effort to control for over parameterisation of models. Bozdogan (1987) extended the theory of AIC to show that it is a measure of the badness of fit minus a measure of complexity of the model. The paper investigated the properties of the AIC and then extended it to a consistent measure.

Bentler Comparative Fit Index

The comparative fit index (CFI) was introduced by Bentler (1990). It is a normed index that is based on comparing the fit functions between two nested models as follows

$$CFI = 1 - \frac{N_k \widetilde{F}^0_k,}{N_I \widetilde{F}^0_I} \tag{4.24}$$

where $\widetilde{F}_k^0$ is the minimum of the fitting function for model M_k and similarly $\widetilde{F}_I^0$ is the minimum of the fitting function for model M_I.

Cutoff Criteria for the Fit Indices

The most recent study of cutoff criteria (Hu and Bentler, 1999) recommends that a cutoff value of 0.95 be used for the CFI, a cutoff value of 0.08 for the standardised RMR and a value of 0.06 for the RMSEA. The NFI and GFI should be above 0.9 (Kline, 1998) and the AIC should be as low as possible. These however, are just rules of thumb and there are no definitive answers for the question of good model fit. These rules of thumbs try to minimise Type I and II errors (Hu and Bentler, 1999).

4.1.3 Normality Issues

West et al. (1995) review the effects of non-normal variables on structural equation modelling. Four main results are presented. The first result, for continuous non-normal variables, is that the maximum likelihood and generalised least squares estimators produce increasingly inflated χ^2 values for increasingly non-normal variables. The second point is that the two estimation methods above produce slightly inflated χ^2 values for small sample sizes, this effect is present even for multivariate normal data. The third effect of non-normal variables is that the fit indices, such as the CFI discussed above, are under-estimated. Finally the standard errors of the parameter estimates are under-estimated.

This, in a sense, makes it harder for a model with non-normal variables to meet the fit index criteria for good model fit. This chapter uses two methods to combat the non-normality issues. Ideally the asymptotically distribution free method estimator (Browne, 1984) would be used over maximum likelihood estimation, however this is not possible for the sample sizes involved in this study. Two other methods will be used instead. The first method is the mean adjusted chi-square developed by (Satorra and Bentler, 1994). The second method used is that of bootstrapping (Efron, 1979). These methods are presented below.

The Mean Adjusted Chi-Square

For a vector of population moments $\boldsymbol{\sigma}$ containing the nonredundant elements of the population covariance matrix $\boldsymbol{\Sigma}$ and a parameter vector $\boldsymbol{\theta}$, the mean adjusted chi-square, with d degrees of freedom, is defined as (Satorra and Bentler, 1994)

$$\chi^2_{adj} = \frac{d}{trace\{\boldsymbol{U}_n\boldsymbol{\Gamma}_n\}}\chi^2, \tag{4.25}$$

where the degrees of freedom, d, is the nearest integer to

$$d' = \frac{(trace\{\boldsymbol{U}_n\boldsymbol{\Gamma}_n\})^2}{trace\{(\boldsymbol{U}_n\boldsymbol{\Gamma}_n)^2\}}. \tag{4.26}$$

$\boldsymbol{\Gamma}_n$ is the estimate of the asymptotic covariance matrix and $\boldsymbol{U}_n$ is the consistent estimate of $\boldsymbol{U}$ where

$$\mathbf{U} = (\mathbf{W}^{-1} - \mathbf{W}^{-1} \triangle (\triangle' \mathbf{W}^{-1} \triangle)^{-1} \triangle' \mathbf{W}^{-1}), \tag{4.27}$$

for some $\boldsymbol{W}$ weight matrix used in the estimation method and $\triangle$ is the Jacobian matrix $(\partial\boldsymbol{\sigma}/\partial\boldsymbol{\theta}')$

Satorra and Bentler (1994) have developed this correction to the test statistic so that the test statistic more closely follows the chi-square distribution and this correction leads to appropriate results for general types of distributions.

Bootstrapping

One method to combat the normality assumptions is to use bootstrapping (Efron, 1979). Bootstrapping is the re-sampling of a dataset with replacement to estimate the sampling distribution by investigating the variation of the statistic within the sample (Mooney and Duval, 1993). The following steps involved in bootstrapping structural equation models are summarised from Yung and Bentler (1996).

- Step 1) Let R be the re-sampling space, which contains the n data points $(x_1, x_2, \ldots, x_n)$.
- Step 2) Draw a sample of m observations randomly from R with replacement. This new sample is used in the structural equation model to obtain the parameter estimates $(\boldsymbol{\Lambda_y}^*, \boldsymbol{\Lambda_x}^*, \boldsymbol{B}^*, \boldsymbol{\Gamma}^*, \boldsymbol{\Phi}^*, \boldsymbol{\Psi}^*, \boldsymbol{\theta}_\epsilon^*, \boldsymbol{\theta}_\delta^*)$ and fit indices (e.g. $\chi^{2*} = (m-1)F_{ML}^*$).
- Step 3) Step 2 is repeated B times to give the set of bootstrapped values for the model parameter estimates and fit indices e.g. $\{\chi_i^{2*}, i = 1, 2, \ldots, B\}$.

The bootstrap percentile confidence interval is given by the $2.5th$ and 97.5 th percentile as 95% of the sample are within the interval i.e.

$$P(t^*_{(\alpha/2)} < t < t^*_{((1-\alpha/2))}) = 0.95. \tag{4.28}$$

for the bootstrap parameter t^*. Further bootstrap theory can found in Efron and Tibshirani (1986), Stine (1990) and Davison and Hinkley (1997). Bootstrapping is used wherever possible, if bootstrapping is not possible then the mean adjusted chi-square is used instead.

The Chi-Square Transformation

Naïve bootstrapping performs poorly when used on the chi-square statistic for structural equation modelling and a transformation can be used to overcome this problem (Bollen and Stine, 1992).

Let T be the test statistic for the original sample and T^* the test statistic from the bootstrap sample. The original sample results in a test statistic that has a noncentral χ^2 distribution and the asymptotic expectation is (Bollen and Stine, 1992)

$$AE(T) = df + \kappa, \tag{4.29}$$

where df is the degrees of freedom and κ is the noncentrality parameter. Similarly the asymptotic variance is given as

$$AVAR(T) = 3df + 4\kappa. \tag{4.30}$$

The noncentrality parameter for the bootstrap sample is equal to the test statistic T (Bollen and Stine, 1992) so that

$$E^* \approx df + T. \tag{4.31}$$

Bollen and Stine (1992) take the expectation with respect to the original population and substitute equation 4.31 to get

$$E[E^*(T^*)] \approx df + df + \kappa \tag{4.32}$$

$$\approx 2df + \kappa. \tag{4.33}$$

Similarly Bollen and Stine (1992) show that under bootstrapping the approximate variance of the test statistic (T^*) is

$$VAR^*(T^*) \approx 2df + 4T, \tag{4.34}$$

thus

$$E[VAR^*(T^*)] \approx 6df + 4\kappa. \tag{4.35}$$

Thus Bollen and Stine (1992) have shown that the mean of T^*, the bootstrap test statistic, exceeds the original test statistic (T) by approximately the degrees of freedom (DF), likewise the variance of the test statistic exceeds that of the original sample. So Bollen and Stine (1992) have shown that the null hypothesis is violated under bootstrapping regardless of whether the null hypothesis is true for the original sample.

To correct this Bollen and Stine (1992) developed the following transformation

$$\mathbf{Z} = \mathbf{Y}\mathbf{S}^{-\frac{1}{2}}\widehat{\Sigma}^{\frac{1}{2}} \tag{4.36}$$

where $\mathbf{Y}$ is the $N \times p$ data matrix of centred observed variables, $\mathbf{S}$ is the sample covariance matrix for the data ($\mathbf{S} = \mathbf{Y}'\mathbf{Y}/(N-1)$ and $\widehat{\Sigma}$) is the estimated implied covariance matrix. $\mathbf{A} = \mathbf{A}^{\frac{1}{2}'}\mathbf{A}^{\frac{1}{2}}$ is the Cholesky factorisation used to obtain the square root of a positive definite matrix.

Bollen and Stine (1992) show that under the null hypothesis the sampling distribution of the modified chi-square behaves approximately as the sampling distribution from the original population. The proof is presented here for convenience.

$$\begin{aligned}
\mathbf{Z}'\mathbf{Z}/(N-1) &= \widehat{\Sigma}^{\frac{1}{2}}\boldsymbol{S}^{-\frac{1}{2}}\boldsymbol{Y}'\boldsymbol{Y}\mathbf{S}^{-\frac{1}{2}}\widehat{\Sigma}^{\frac{1}{2}}/(N-1) && (4.37)\\
&= \widehat{\Sigma}^{\frac{1}{2}}\boldsymbol{S}^{-\frac{1}{2}}\mathbf{S}\mathbf{S}^{-\frac{1}{2}}\widehat{\Sigma}^{\frac{1}{2}} && (4.38)\\
&= \widehat{\Sigma}^{\frac{1}{2}}\boldsymbol{S}^{-\frac{1}{2}}\mathbf{S}^{\frac{1}{2}}\mathbf{S}^{\frac{1}{2}}\mathbf{S}^{-\frac{1}{2}}\widehat{\Sigma}^{\frac{1}{2}} && (4.39)\\
&= \widehat{\Sigma}^{\frac{1}{2}}\widehat{\Sigma}^{\frac{1}{2}} && (4.40)\\
&= \widehat{\Sigma} && (4.41)
\end{aligned}$$

So the covariance matrix is $\widehat{\Sigma}$ as required. The bootstrap is performed by taking samples from $\mathbf{Z}$ rather than the original data. This leads to a modified bootstrap test statistics T_m^* and the means and variances for this modified test statistic are (Bollen and Stine, 1992)

$$\begin{aligned}
E^*(T_m^*) &\approx df && (4.42)\\
E[E^*(T_m^*)] &\approx df, && (4.43)
\end{aligned}$$

and

$$\begin{aligned}
VAR^*(T_m^*) &\approx 2df && (4.44)\\
E[VAR^*(T_m^*)] &\approx 2df. && (4.45)
\end{aligned}$$

Thus bootstrapping on the modified data leads to a test statistic that behaves like the sampling distribution of the original population, under the null hypothesis. This transformation has been used for the bootstrapping in the following sections and was calculated within Matlab (Mathworks Inc). The bootstrapping is performed on the entire sample (both confirmatory and exploratory) as the bootstrapping is performed after the confirmatory analysis.

4.2 Confirmatory Factor Analysis

Chapter 3 introduced a number of models to best describe the underlying component structure of the personality traits and symptom scores separately. Due to the lack of agreement in methods for choosing the number of components to rotate, models were developed with varying numbers of components. Principal component (PC), independent component (IC) and factor analysis (FA) methods were used to investigate the component structures. Confirmatory factor analysis (CFA) will now be used to decide the best model for the males and females at baseline and after treatment separately, for the personality and symptom scores.

The CFA models use a small number of variables, in that TCI has seven variables and SCL has nine, so latent factors are often described by a single indicator variable, thus causing identification issues. The general rule of thumb is that three variables are usually needed per latent construct to guarantee identification (Bollen, 1989). To overcome this the following procedure was used. This procedure is described fully in Bollen (1989).

- Step 1: Run models on exploratory data to find R^2 values as an indicator of reliability for each single indicator variable.
- Step 2: In the confirmatory models set the regression coefficients for the single indicators to one and set the error variance equal to $(1 - R^2) \times variance$ of the variable.
- Step 3: Run the CFA models.

The CFA results will be reported separately for personality and then symptoms.

4.2.1 Results for Personality

The results from the Flury test in Chapter 3 suggested that the males and females should be treated separately for personality at both time points. The CFA models reflect this with males and females kept separate. Any gender differences or similarities will be investigated further by multigroup analysis (Everitt, 1984) and changes across time analysed using longitudinal analysis (Everitt, 1984).

The Females at Baseline

There were nine different personality models to analyse for the depressed females at baseline. Table 4.1 presents the fit index results for each of the models. The indices that meet the appropriate criteria, as discussed in Section 4.1.2, are presented in bold. If all the fit indices are in bold then the model has a reasonable fit. The different models are

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/IC	**0.940**	0.698	**0.004**	0.229	0.851	10.60	0.838
3PCs/FAs	0.899	0.646	**0.002**	0.215	0.744	27.29	0.729
4 PCs	0.768	0.349	0.204	0.520	-1.377	314.61	-1.124
5 PCs	0.780	0.383	0.464	0.596	-2.124	416.63	-1.124
3 ICs	0.756	0.358	0.231	0.570	-1.399	304.21	-1.206
4 ICs	0.795	0.521	0.331	0.534	-2.010	399.04	-1.685
5 ICs	0.749	0.297	0.474	0.621	-2.390	452.95	-2.002
1 FA	**0.985**	**0.926**	**0.002**	**0.086**	**0.955**	**-0.21**	**0.916**
4 FA	0.761	0.329	0.192	0.514	-1.323	307.21	-1.077

Table 4.1: The depressed females TCI models at baseline using the confirmatory dataset. (Key: 1PC = Principal component model with 1 component; 3PCs = Principal component model with 3 components.)

presented down the rows with the fit indices in the columns. For the females at baseline the PC and IC one component solution lead to the same model and as well as the three component PC and FA models.

The one component factor analysis model (bolded) is the only model to meet all fit index criteria and this model is presented in Figure 4.2. The entire sample was used (exploratory and confirmatory) for the bootstrapping, as the confirmatory analysis had already been conducted. Combining the samples increases the power for investigating the fit of the model. The loadings presented are unstandardised hence appear low. The 90% confidence intervals on all the factor loadings do not include zero indicating that all the loadings are significant.

The model describes one latent factor that leads to the four manifest TCI traits. The first trait, harm avoidance, has a high positive standardised loading and a small error variance, indicating that it is a good indicator of the underlying latent construct. Harm avoidance is contrasted against persistence, self directedness and cooperativeness. Persistence has the smallest loading and highest error variance making it the least reliable indicator for the underlying personality factor. The error variances are all proper solutions however one confidence interval includes zero (the third error variance), thus this error variance is not significantly different from zero.

The bootstrapped fit indices are presented in Table 4.2. Under naïve bootstrapping techniques the GFI and RMR have confidence intervals in the right bounds indicating good model fit. However the other fit indices have an upper bound in the right interval

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.979	(0.955, 0.994)	0.997	(0.988, 1.000)
AGFI	0.894	(0.776, 0.969)	0.986	(0.941, 0.999)
RMR	0.002	(0.001, 0.003)	0.001	(0.000, 0.001)
Chi-Square	9.994	(2.762, 22.069)	1.216	(0.081, 5.286)
df	2		2	
p-value	0.007	(0.251, 0.000)	0.545	(0.960, 0.071)
RMSEA	0.134	(0.042, 0.213)	0.000	(0.000, 0.086)
CFI	0.942	(0.864, 0.994)	1.000	(0.975, 1.000)
AIC	5.994	(-1.238, 18.069)	-2.784	(-3.919, 1.286)
NFI	0.931	(0.855, 0.980)	0.991	(0.961, 0.999)

Table 4.2: The bootstrapped fit indices for the female's baseline TCI model.

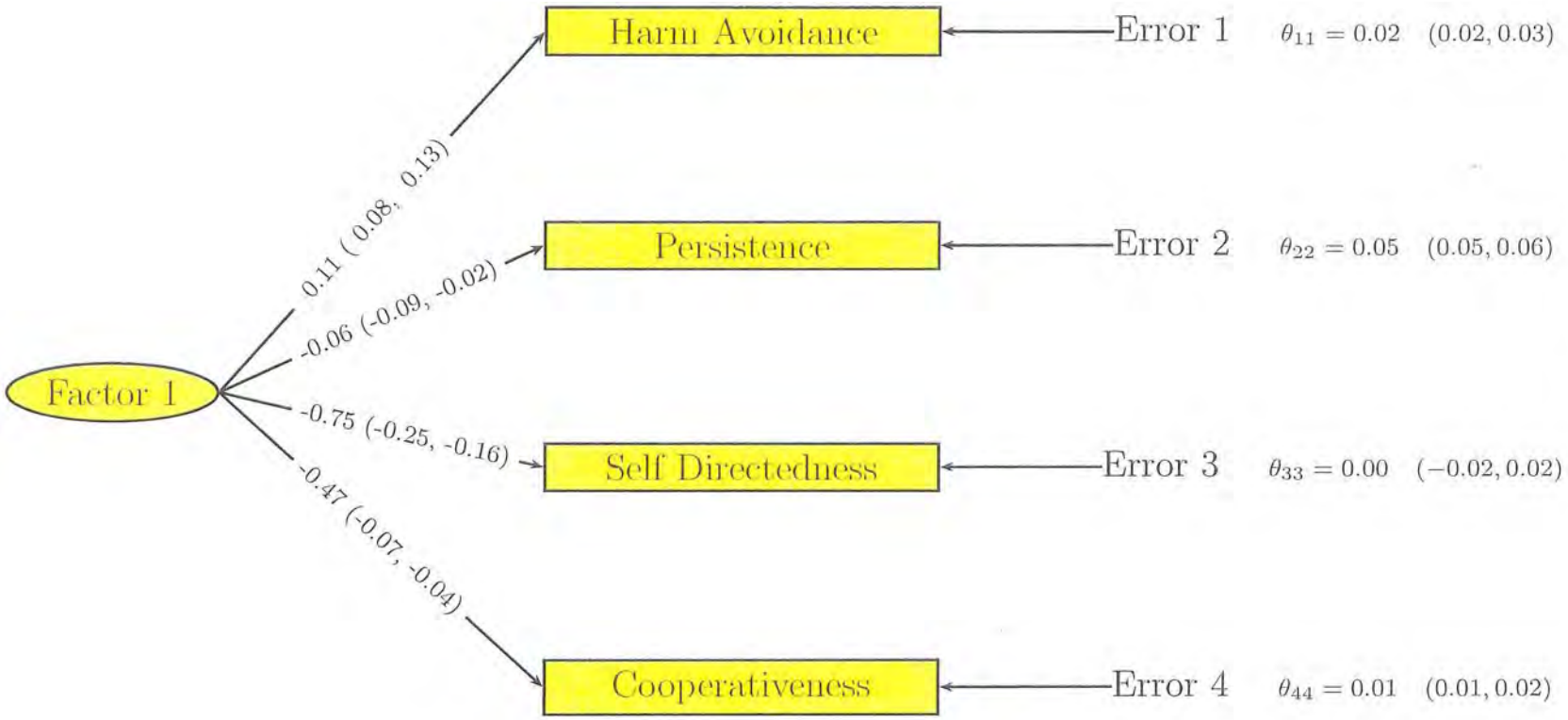


Figure 4.2: The depressed females structural model of TCI at baseline.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/FA	**0.969**	0.843	**0.065**	0.140	0.933	0.579	0.897
3 PCs	0.878	0.737	0.110	0.160	0.640	8.869	0.574
5 PCs	**0.914**	0.800	0.092	0.125	0.798	0.297	0.703
1 IC	**0.989**	**0.945**	**0.039**	**0.000**	**1.000**	**-2.493**	**0.952**
3 ICs	0.897	0.778	0.118	0.136	0.740	2.803	0.648
5 ICs	0.895	0.706	0.141	0.183	0.637	12.093	0.608
3 FA	**0.908**	0.678	0.105	0.198	0.718	9.537	0.693
4 FA	**0.914**	0.758	0.087	0.141	0.783	3.191	0.717

Table 4.3: The depressed females TCI models after treatment, using the confirmatory dataset. (Key: 1PC = Principal component model with 1 component; 3PCs = Principal component model with 3 components.)

and a lower bound that is not, indicating that the sample size may be too small for a definitive answer on model fit.

However Bollen and Stine (1992) suggested that, under the so called naïve bootstrapping, the fit indices would be miscalculated (see Section 4.1.3). Using their suggested transformation, bootstrap results are presented in the Table 4.2. The confidence intervals of all the indices are in the right bounds, though the RMSEA upper estimate of 0.086 may be problematic. The chi-square interval is large, but both lower and upper bounds are non significant suggesting good model fit. This would suggest that under bootstrapping the sample size is sufficient to conclude that the model of 1 FA is appropriate for explaining the underlying personality structure for the females at baseline.

The Females After Treatment

After treatment the independent component model with one component is the best fitting model (see bolded model Table 4.3). Figure 4.3 presents this one component model, bootstrapped on the entire sample (both exploratory and confirmatory). The model has a single underlying factor that has a positive loading on harm avoidance and negative loadings on persistence, self directedness and self transcendence. This post treatment model differs from the corresponding baseline model, with self transcendence replacing cooperativeness. Self directedness has the highest loading and smallest error variance (Figure 4.3). The confidence intervals are all nonzero indicating significant nonzero loadings and error variances (Figure 4.3).

The bootstrapped fit index results are presented in Table 4.4. Most of the fit indices

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.991	(0.965, 0.999)	0.995	(0.979, 1.000)
AGFI	0.954	(0.824 , 0.996)	0.975	(0.893, 0.998)
RMR	0.002	(0.000 , 0.003)	0.001	(0.000, 0.002)
Chi-Square	2.538	(0.211, 10.397)	1.364	(0.101, 5.986)
df	2		2	
p-value	0.281	(0.900, 0.006)	0.506	(0.951 , 0.0501)
RMSEA	0.045	(0.000, 0.177)	0.000	(0.000 , 0.122)
CFI	0.991	(0.869, 1.000)	1.000	(0.934 , 1.000)
AIC	-1.462	(-3.789, 6.397)	-2.636	(-3.899 , 1.986)
NFI	0.960	(0.849, 0.997)	0.978	(0.908 , 0.998)

Table 4.4: The bootstrapped fit indices for the after treatment female TCI model.

have confidence intervals in the correct bounds. The lower bound of the comparative fit index is 0.934, which using the criteria of 0.95 or more for that fit index, would make this interval inconclusive. The chi-square interval is again large and the lower bound of 0.0501 is only just not significant. This would suggest that ideally, a larger sample would be needed to be sure that the model is a good fit. Generally the model fits well and is a good representative of the underlying personality structure post treatment (Figure 4.3).

The Males at Baseline

The males at baseline have only poor fitting models (Table 4.5). The PCA, ICA and FA methods are all unsuccessful at producing a good general male baseline model. There could be a number of reasons for this poor fit. One being that the models presented have only allowed manifest variables to load on one latent variable. In reality the true personality model may be more complex, loading on multiple latent variables. The PCA, ICA and FA methods appear to have found specific models that do not generalise well to the confirmatory data for the males at baseline. No models will be retained for further analysis.

The Males After Treatment

Post treatment model results are presented in Table 4.6 and the one component model (bolded) is the best. Figure 4.4 represents the one component model for the depressed males after treatment. This model has the same form as the depressed females baseline model in that harm avoidance, persistence, self directedness and cooperativeness are

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC	0.866	0.597	0.107	0.213	0.839	5.179	0.793
2 PCs/ICs	0.864	0.643	0.104	0.176	0.824	3.122	0.756
5 PCs	0.846	0.641	0.091	0.204	0.746	10.480	0.685
1 IC	**0.916**	0.579	0.109	0.270	0.878	4.580	0.856
5 ICs	**0.903**	0.729	0.117	0.153	0.880	**0.596**	0.812
1 FA	**0.933**	0.663	0.110	0.230	0.899	2.761	0.873
2 FAs	0.858	0.467	0.188	0.310	0.766	13.3127	0.747
4 FAs	0.8356	0.616	0.097	0.209	0.732	11.682	0.674

Table 4.5: The depressed males TCI models at baseline.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/IC/FA	**0.992**	**0.961**	**0.023**	**0.000**	**1.000**	**-3.292**	**0.987**
2 PCs	0.880	0.686	0.100	0.170	0.835	2.429	0.764
3 PCs/ICs	0.893	0.718	0.091	0.158	0.859	0.931	0.784
5 PCs/FAs	0.846	0.641	0.091	0.204	0.746	10.479	0.685
2 ICs	**0.913**	0.673	0.103	0.211	0.852	4.011	0.812
5 ICs	0.870	0.636	0.136	0.204	0.789	8.659	0.738
2 FAs	0.754	0.386	0.257	0.702	-1.014	126.963	-0.828
3 FAs	**0.903**	0.753	**0.005**	0.152	0.835	0.390	0.751

Table 4.6: The depressed males TCI models after treatment.

included. Self directedness has the strongest loading on the underlying factor and the smallest error variance. Harm avoidance is contrasted against persistence, self directedness and cooperativeness. All the loadings are significantly different from zero as the confidence intervals do not contain zero. One of the error variances (the third one) has a confidence interval that includes zero suggesting that the error variance is not significantly different from zero.

The bootstrapped fit indices are presented in Table 4.7. All the fit indices are in the appropriate bounds when using the Bollen Stine transformation, apart from the RMSEA estimate. The chi-square interval is large and the lower bound p-value is 0.056, thus not significant. The post treatment model (Figure 4.4) has a good fit and appears to model the underlying personality structure for the males after treatment. It also appears to mirror the females baseline personality model.

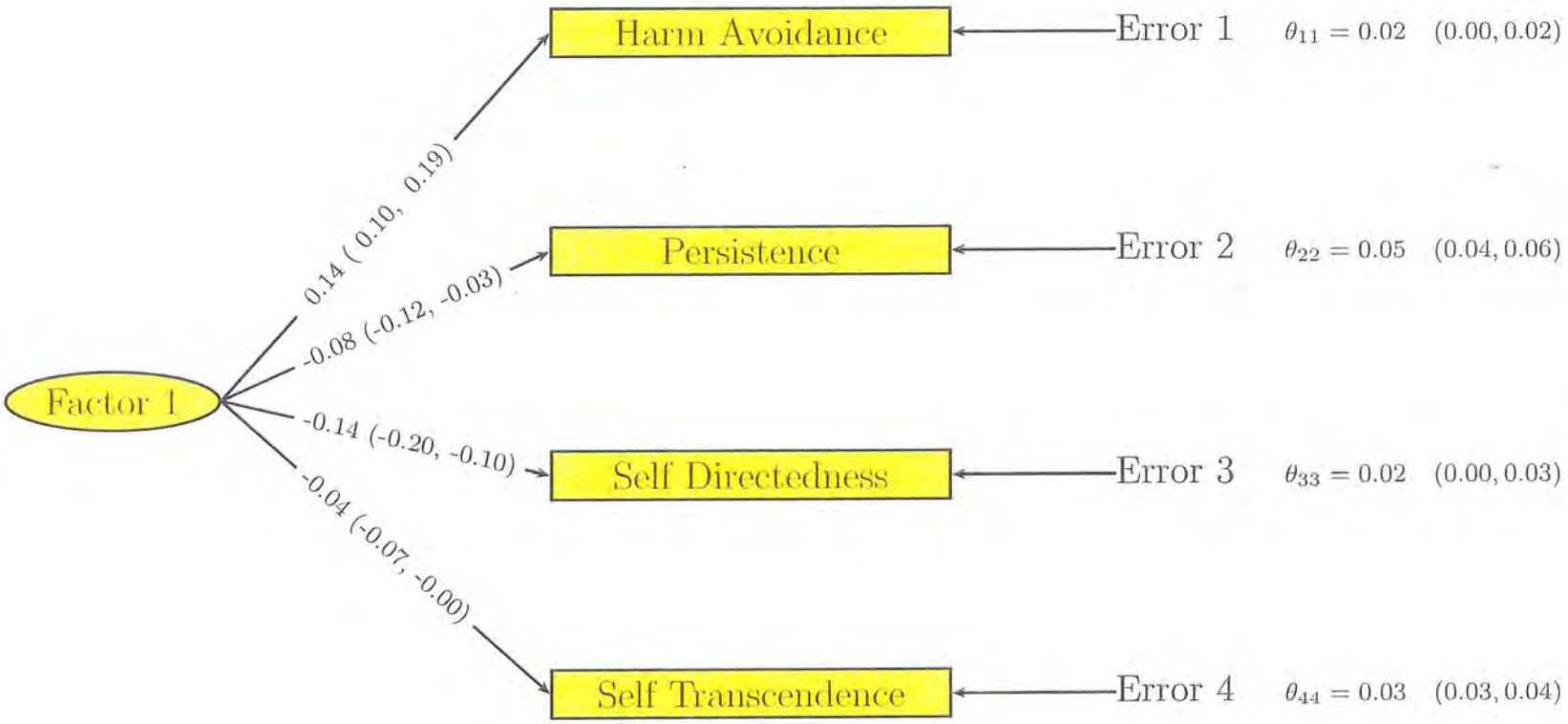


Figure 4.3: The depressed females structural model of TCI after treatment.

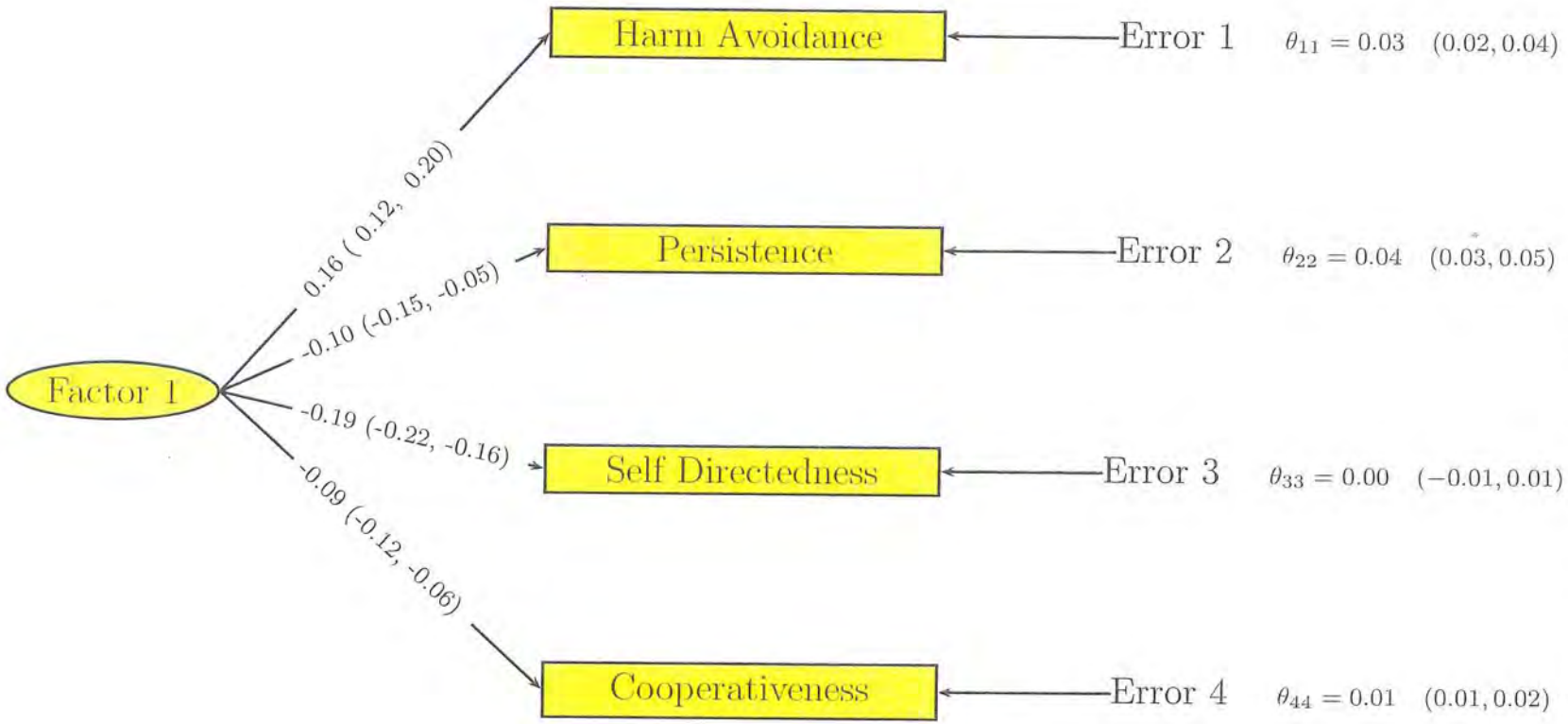


Figure 4.4: The depressed males structural model of TCI after treatment.

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90%CI)	Median	(90% CI)
GFI	0.986	(0.947, 0.999)	0.990	(0.960, 0.999)
AGFI	0.928	(0.736, 0.994)	0.950	(0.798, 0.996)
RMR	0.001	(0.000, 0.003)	0.001	(0.000, 0.003)
Chi-Square	2.027	(0.149, 8.046)	1.348	(0.103, 5.774)
df	2		2	
p-value	0.363	(0.928, 0.018)	0.510	(0.950, 0.056)
RMSEA	0.014	(0.000, 0.212)	0.000	(0.000, 0.168)
CFI	1.000	(0.926, 1.000)	1.000	(0.954, 1.000)
AIC	-1.973	(-3.851, 4.046)	-2.652	(-3.897, 1.774)
NFI	0.976	(0.906, 0.998)	0.984	(0.932, 0.999)

Table 4.7: The bootstrapped fit indices for the males post treatment TCI model.

4.2.2 Summary of the personality models

For personality only models involving one component were successful in describing the latent structure. Thus the Cloninger personality model has been reduced to one latent variable that leads to four manifest TCI variables. The models all contain harm avoidance in contrast with persistence and self directedness. Cooperativeness and self transcendence were time specific for the females.

For the females, at baseline factor analysis calculated the best model, however after treatment the IC method produced the best model. For the males after treatment, the three methods, PCA, ICA and FA, produced the same one component model and this was found to be the best model.

4.2.3 Results for the Symptoms

The Males and Females at Baseline

Using the results of the Flury test in Chapter 3, males and females were combined for the symptom models at baseline. The CFA results are presented in Table 4.8 with the six principal components as the best model (bolded).

Figure 4.5 represents the baseline symptoms model with bootstrapped parameter estimates based on the whole sample. Unstandardised solutions are presented for the factors apart from those with single indicators, where the loading has been preset to one and the variance set from the reliability and variance of the exploratory sample (see Section 4.2).

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/IC/FA	0.863	0.771	**0.052**	0.133	0.915	52.663	0.890
2 PCs	0.885	0.801	**0.050**	0.125	0.928	41.716	**0.904**
4 PCs	0.891	0.787	**0.048**	0.133	0.928	44.468	**0.907**
6 PCs	**0.927**	0.837	**0.041**	0.109	**0.958**	**19.472**	**0.939**
2 ICs	0.892	0.799	**0.053**	0.144	0.921	36.311	**0.901**
4 ICs	**0.921**	0.822	**0.046**	0.128	0.944	27.370	**0.926**
6 ICs	**0.905**	0.787	**0.049**	0.150	0.923	43.830	**0.905**
2 FAs	0.8818	0.795	**0.033**	0.128	0.899	50.325	0.871
4 FAs	**0.904**	0.794	**0.033**	0.126	0.921	38.609	0.898

Table 4.8: The depressed males and females combined SCL models at baseline, using the confirmatory dataset.

Covariance	Estimate	(90% CI)
Factor 1 Factor 2	0.861	(0.805, 0.904)
Factor 1 Factor 3	0.871	(0.846, 0.894)
Factor 2 Factor 3	0.824	(0.787, 0.856)
Factor 1 Factor 4	0.814	(0.768, 0.855)
Factor 2 Factor 4	0.818	(0.775, 0.856)
Factor 3 Factor 4	0.695	(0.648, 0.735)
Factor 1 Factor 5	0.839	(0.806, 0.866)
Factor 2 Factor 5	0.719	(0.663, 0.764)
Factor 3 Factor 5	0.768	(0.735, 0.797)
Factor 4 Factor 5	0.660	(0.606, 0.710)
Factor 1 Factor 6	0.645	(0.574, 0.705)
Factor 2 Factor 6	0.667	(0.593, 0.730)
Factor 3 Factor 6	0.595	(0.529, 0.653)
Factor 4 Factor 6	0.540	(0.458, 0.613)
Factor 5 Factor 6	0.558	(0.491, 0.618)

Table 4.9: The covariance estimates for the males and females baseline SCL model.

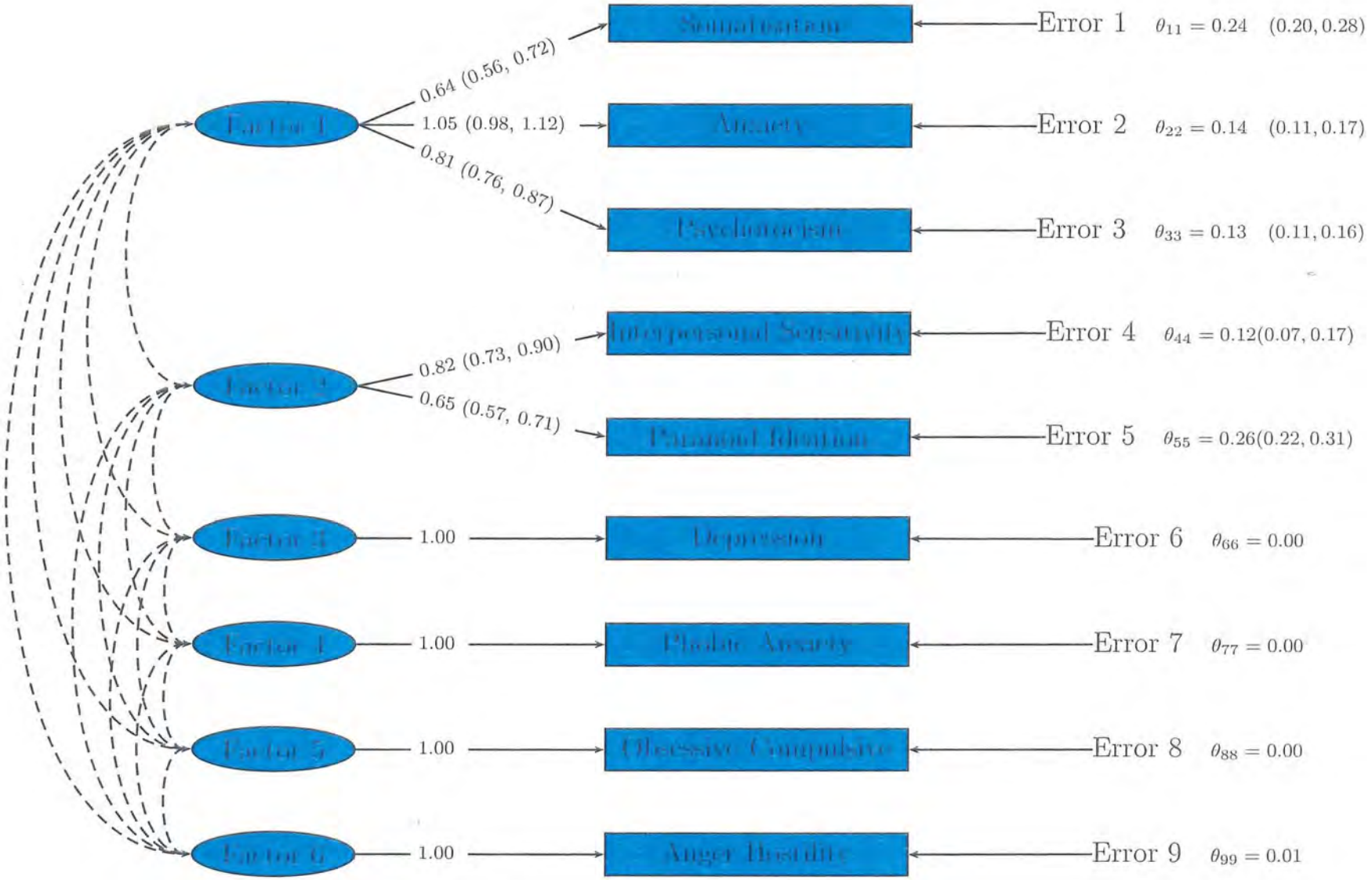


Figure 4.5: The depressed males and females symptom model at baseline.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.881	(0.858, 0.903)	0.986	(0.977, 0.992)
AGFI	0.733	(0.681, 0.781)	0.968	(0.948, 0.983)
RMR	0.336	(0.289, 0.385)	0.043	(0.018, 0.102)
Chi-Square	214.568	(173.157, 263.004)	22.659	(12.167, 37.428)
df	20		20	
p-value	0.000	(0.000, 0.000)	0.306	(0.910, 0.0104)
RMSEA	0.168	(0.149, 0.188)	0.020	(0.000, 0.050)
CFI	0.890	(0.855, 0.917)	0.999	(0.994, 1.000)
AIC	174.568	(133.157, 223.004)	-17.341	(-27.833, -2.572)
NFI	0.881	(0.846, 0.908)	0.992	(0.986, 0.996)

Table 4.10: The bootstrapped fit indices for the males and females baseline SCL model.

Table 4.9 presents the covariances between the latent symptom factors. All the confidence intervals are positive suggesting the symptom factors are all positively interrelated. Table 4.10 presents the bootstrapped fit indices. The chi-square is the only fit index outside the good fit boundaries. As has been previously observed the chi-square has a large confidence interval, but in this case, its lower and upper limit has p-values of 0.01 to 0.91 implying that the sample is not large enough for a definitive answer on model fit. The other fit indices all suggest that the model is a good representation of the latent symptom structure at baseline.

The Females After Treatment

The post treatment results are presented in Table 4.11 for the females. The table shows that no reasonably well fitting models were found for the post treatment symptoms. Due to the normality issues the fit indices may be adversely affected so the female's 2 IC solution will be retained for bootstrapping as the fit indices are near the recommended levels and the normality issues may be resolved by bootstrapping.

Figure 4.6 represents the symptom model for the depressed females after treatment. The factor loadings are all positive and significantly different from zero, this is evidenced by positive, non zero confidence intervals. The covariance between the two factors is large and positive, suggesting that the symptoms are all positively interrelated, so a person high on one symptom is likely to be high on the other symptoms in the model. All, except one error variance, are significantly different from zero. These error variances are small and have positive non zero confidence intervals.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/IC/FA	0.767	0.611	**0.070**	0.211	0.865	81.842	0.839
2 PCs/FAs	0.782	0.623	**0.071**	0.198	0.884	67.127	0.858
3 PCs	0.830	0.681	**0.052**	0.179	0.913	46.109	0.888
4 PCs	0.784	0.577	**0.071**	0.209	0.887	68.331	0.864
5 PCs/FAs	0.825	0.658	**0.063**	0.189	0.908	51.409	0.884
2 ICs	0.877	0.734	**0.049**	0.163	0.946	**18.500**	**0.926**
3 ICs	0.823	0.664	**0.054**	0.190	0.910	43.167	0.888
4 ICs	0.793	0.608	**0.076**	0.223	0.877	66.577	0.855
5 ICs	0.851	0.664	**0.063**	0.201	0.915	42.839	0.896
3 FAs	0.791	0.608	**0.072**	0.201	0.890	64.513	0.866

Table 4.11: The depressed females SCL models after treatment.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.881	(0.817, 0.932)	0.957	(0.919, 0.981)
AGFI	0.743	(0.606, 0.854)	0.908	(0.825, 0.959)
RMR	0.012	(0.008, 0.017)	0.007	(0.004, 0.010)
Chi-Square	77.198	(42.407, 128.366)	25.987	(11.125, 52.336)
df	13		13	
p-value	0.000	(0.000, 0.000)	0.017	(0.600, 0.000)
RMSEA	0.176	(0.119, 0.236)	0.079	(0.000, 0.138)
CFI	0.933	(0.887, 0.968)	0.986	(0.958, 1.000)
AIC	51.198	(16.407, 102.366)	-0.013	(-14.875, 26.336)
NFI	0.921	(0.876, 0.954)	0.972	(0.945, 0.988)

Table 4.12: The bootstrapped fit indices for the females after treatment SCL model.

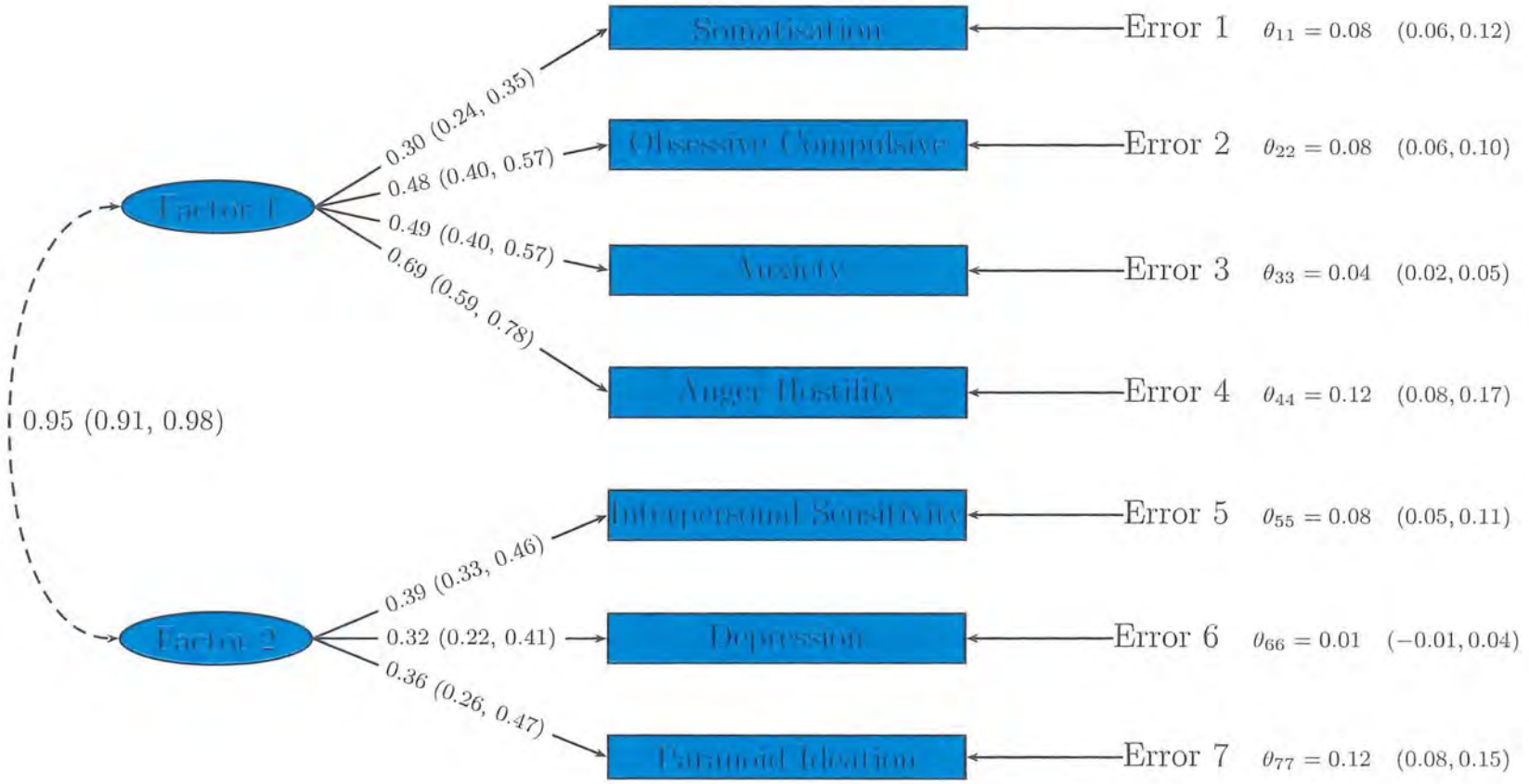


Figure 4.6: The depressed females SCL model post treatment.

Confirmatory Dataset

Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/IC/FA	0.605	0.341	0.093	0.299	0.716	50.280	0.661
3 PCs	0.611	0.327	0.094	0.306	0.712	52.121	0.662
4 PCs	0.721	0.455	0.096	0.262	0.814	27.460	0.761
5 PCs	0.745	0.454	**0.068**	0.227	0.872	13.750	0.819
6 PCs	0.745	0.501	0.081	0.236	0.849	17.926	0.792
3 ICs	0.663	0.361	0.102	0.309	0.758	38.957	0.713
4 ICs	0.681	0.316	0.101	0.301	0.775	40.021	0.733
5 ICs	0.640	0.295	0.152	0.316	0.729	20.701	0.686
6 ICs	0.687	0.328	0.095	0.283	0.802	32.700	0.757
3 FAs	0.652	0.398	0.097	0.271	0.776	34.933	0.717
4 FAs	0.731	0.424	0.084	0.243	0.854	18.715	0.803
5 FAs	0.756	0.523	0.069	0.226	0.861	**14.736**	0.803

Table 4.13: The depressed males symptom models at after treatment.

Table 4.12 presents the bootstrapped fit indices for the post treatment female SCL model. Most of the fit indices are in the appropriate bounds, however some of the confidence intervals are ambiguous. The chi-square interval is particularly large, probably due to the small sample size. The chi-square interval goes from a p-value of 0.00 to 0.60 suggesting a larger sample is needed. The RMSEA estimate is also ambiguous. However all the other fit indices suggest that the model fit is good. Ideally a larger sample would be used for further testing but this is unavailable for this current study.

The Males After Treatment

The post treatment results are presented in Table 4.13 for the males. The table shows that no reasonably well fitting models were found for the male's post treatment symptoms. No models are retained for further analysis.

4.2.4 Summary of the Symptom Models

All the models exhibit positive loadings of the symptoms and positive covariances between the latent factors suggesting that the symptoms are working in the same direction. In other words, if a person is high on one symptom, the model suggests that he or she will be high on the other symptoms. At baseline more factors were required for an adequate model fit than needed after treatment. This suggests that at baseline, when the patients are suffering from depression, their symptom structure is more complicated than after

treatment. Interestingly the extra factors at baseline are all single indicators, suggesting that these symptoms are distinct symptoms, not part of an overall distress symptom as some of the literature suggests. Studies by Carpenter and Hittner (1995), Bonynge (1993) and Bernstein et al. (1994) present evidence for a single overall factor of general distress, rather than the original nine symptoms. However other studies (Vassend and Skrondal, 1999; Schwarzwald et al., 1991) have presented evidence for more than one factor. Our study suggests that when patients are more severely ill more factors are required to adequately describe the structure of the symptoms, after treatment when the symptoms have declined less factors are needed to adequately describe the symptom structure. This may be one reason why there are discrepancies in the literature, as the studies may have used patients with different degrees of illness severity as well as different types of mental illness.

4.2.5 Combining Personality and Symptoms

To investigate the covariance structure within and between symptoms and personality, combined symptom and personality models were developed using the PCA, ICA and FA methods in Chapter 3 and these models are tested using the confirmatory framework in Appendix B. The three models presented show that symptoms dominate over personality. Generally personality variables did not appear in the models and when they did they had poor loadings. The latent factors that these variables appeared on did not significantly covary with the other factors. This leads us to the conclusion that personality and symptom structures are quite distinct and that the models developed for personality and symptoms separately are more informative.

4.3 Multi-group Analysis

In the previous section the best models were found for each group using confirmatory factor analysis. These models will now be further investigated with the purpose of looking into differences and similarities across gender. In SEM there may be more than one population of interest. Multi-group analysis is used to compare the same model in the different populations or groups (Everitt, 1984; Bollen, 1989). Multi-group analysis investigates a series of hypotheses with increasing strictness of commonality between the groups (Bollen, 1989).

The parameter matrices for groups $g = 1, \ldots, G$ are $\mathbf{\Lambda_x}^{(g)}$, $\mathbf{\Lambda_y}^{(g)}$, $\boldsymbol{B}^{(g)}$, $\mathbf{\Gamma}^{(g)}$, $\mathbf{\Phi}^{(g)}$, $\mathbf{\Psi}^{(g)}$, $\boldsymbol{\theta}_{\epsilon}^{(g)}$ and $\boldsymbol{\theta}_{\delta}^{(g)}$. In a multigroup analysis the first, least restrictive test used is to test for the common form of the structural model. This allows the parameters to be different across the groups. The null hypothesis is H_{form}: same form (same dimensions and same

patterns of fixed, free, and constrained elements in Λ_x, θ_δ and Φ) (Bollen, 1989). Figure 4.7 shows the path diagram for a test of common form across groups 1 and 2. If the chi-squared statistic for this model is non-significant, this tells us that there are no significant differences between the two structural models across the groups.

Further investigation will indicate whether the model has the same parameter estimates across the groups. The first restriction placed on the model is for the regression elements to be the same across the two groups. The null hypothesis is $H_{\Lambda_x}: \Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}$ (Bollen, 1989). This is shown in Figure 4.8. Again a non-significant chi-square suggests that there are no significant differences between the groups under those conditions.

The next step is to test for commonality of the error variances ($H_{\Lambda_x\theta_\delta}: \Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}\ \theta_\delta^{(1)} = \theta_\delta^{(2)}$ (Bollen, 1989)) and finally commonality of the covariances or regressions between the latent variables ($H_{\Lambda_x\theta_\delta\Phi}: \Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}\ \theta_\delta^{(1)} = \theta_\delta^{(2)}\ \Phi^{(1)} = \Phi^{(2)}$ (Bollen, 1989)). This is the most restricted model, for the type of analysis used in this thesis, with all parameters constrained across the groups. Once again a non-significant chi-squared statistic indicates that the groups are not significantly different and can be described by the same model.

Mathematically the model is investigating how close the implied group covariance matrix fits each group sample covariance matrix. When the groups sample covariance matrices are close to the implied covariance structure the model will have an acceptable fit. When the groups are significantly different from the implied structure it will show up as an unacceptable fit for the model. The equation for the Maximum Likelihood fit function in multigroup analysis is (Bollen, 1989)

$$F_{gML} = \log|\Sigma_g| + tr(S_g\Sigma_g^{-1}) - \log|S_g| - (p+q). \tag{4.46}$$

In summary the testing hierarchy is

H_{form}:	same form (same dimensions and same patterns of fixed, free, and constrained elements in Λ_x, θ_δ and Φ)
H_{Λ_x}:	$\Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}$
$H_{\Lambda_x\theta_\delta}$:	$\Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}\ \theta_\delta^{(1)} = \theta_\delta^{(2)}$
$H_{\Lambda_x\theta_\delta\Phi}$:	$\Lambda_x{}^{(1)} = \Lambda_x{}^{(2)}\ \theta_\delta^{(1)} = \theta_\delta^{(2)}\ \Phi^{(1)} = \Phi^{(2)}$.

In other words the model is tested for common form, common latent to manifest regression coefficients, common error variances and finally all common parameters. If, at any stage, the groups are found to be significantly different, the process is stopped.

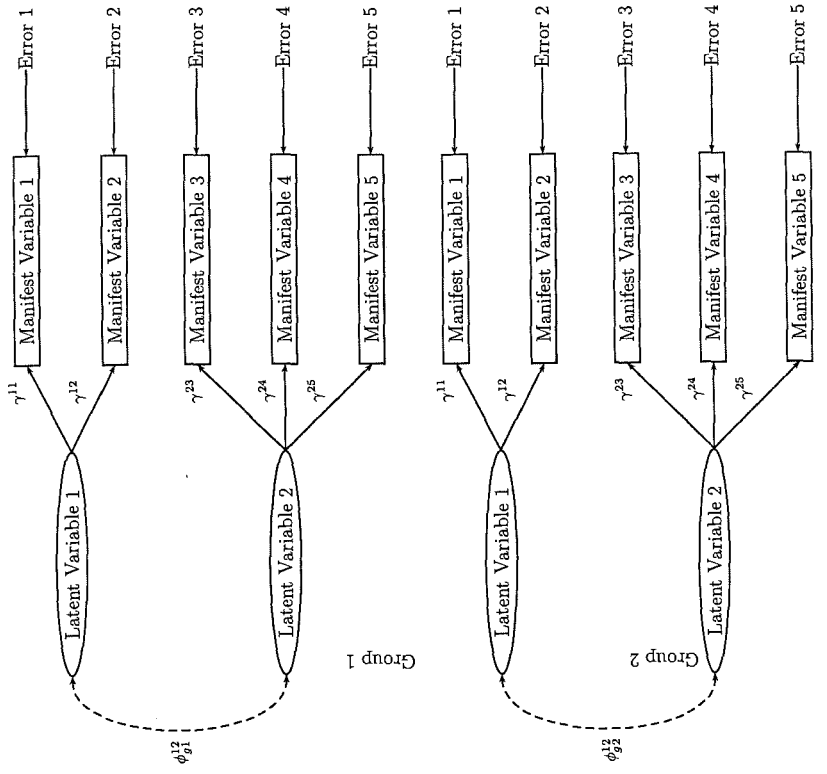


Figure 4.8: The multigroup test for common form and common regression coefficients.

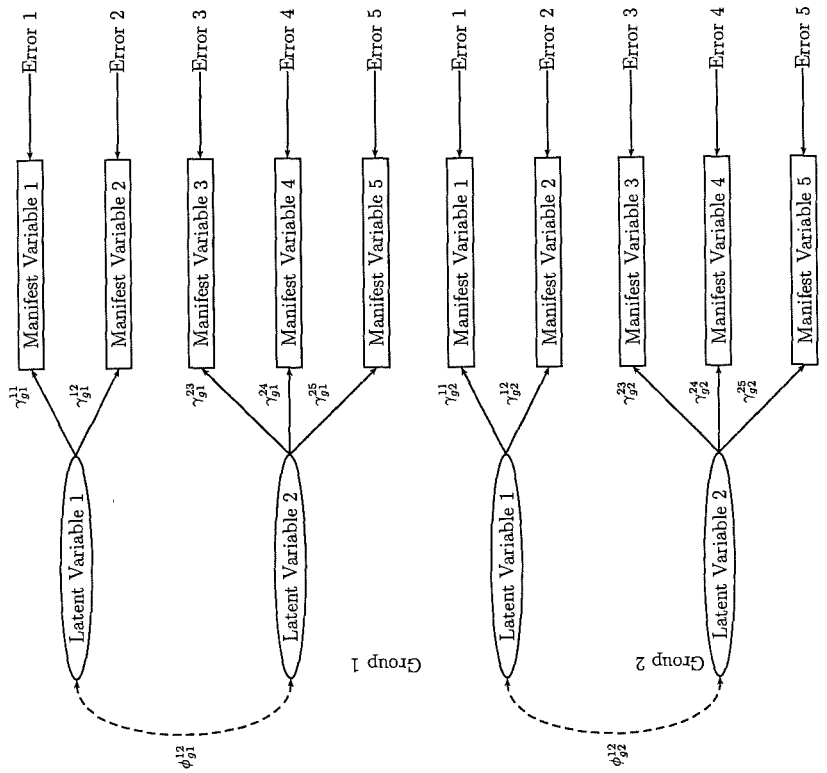


Figure 4.7: The multigroup test for common form.

4.3.1 Multigroup Analysis Results

Using multi-group analysis the differences and similarities between the depressed males and females are investigated. Using the models found from the CFA analysis (Section 4.2), the females are tested on the male structure and vice versa. The model is then tested for common form, common latent to manifest regression coefficients, common error variances and finally all common parameters, if the process is not stopped due to significant differences.

The exploratory and confirmatory data are grouped together to give more data for the test as the cross validation has already been performed.

Personality at Baseline

For the baseline personality the males do not have a reasonable structure (see Table 4.5), so the females structure will be tested across males and females. Table 4.14 presents the multigroup results. The first row presents the maximum likelihood chi-square which has a significant p-value. The second row presents the mean adjusted maximum likelihood chi-square calculated in Mplus (Muthén and Muthén, Version 2, www.statmodel.com), which is also significant ($p = 0.00$). The third and fourth rows are the bootstrap results from both naïve bootstrapping and Bollen-Stine transformed calculated in Statistica's SEPATH (StatSoft, Inc). The first three measures agree that there are significant differences between males and females on baseline personality. The Bollen-Stine transformed chi-square of 3.93 is non-significant however the confidence interval is ambiguous as its lower bound is significant and its upper bound is not significant.

The ambiguous chi-square confidence interval means a definite answer to reject or not can not be made. An investigation was made into the feasibility of doing more bootstrap runs. There may be no point in running ten times as many bootstrap samples as the confidence interval may be appropriate for the original sample size and thus the original sample size may be the limiting factor on the degree of confidence for the bootstrap chi-square.

Figure 4.9 presents the mean and 95% confidence interval for the chi square statistic after each bootstrap run. The flat line is the rejection region for a 5% significance level for the appropriate degrees of freedom. Clearly the mean and lower bound of the confidence interval are below the rejection line, however the upper confidence limit is above the rejection line. This leads to an ambiguity in the test as the confidence interval includes 0.05. Further boot strap runs, say up to 10 000, do not appear to be worthwhile as the interval does not appear to change after the first 2000 runs. This suggests that because of the sample size involved the confidence interval will remain relatively large despite repeated bootstrap runs. A larger sample size is needed.

Method	Hypothesis	χ^2	df	P-value
SEPATH ML	H_{form}	10.38	4	0.03
MPlus MLM	H_{form}	48.91	7	0.00
Naïve bootstrap	H_{form}	14.12 (3.99, 29.38)	4	0.01 (0.00, 0.41)
Bollen-Stine transformed	H_{form}	3.93 (0.51, 10.81)	4	0.42 (0.03, 0.97)

Table 4.14: The multigroup hypothesis test results for personality at baseline.

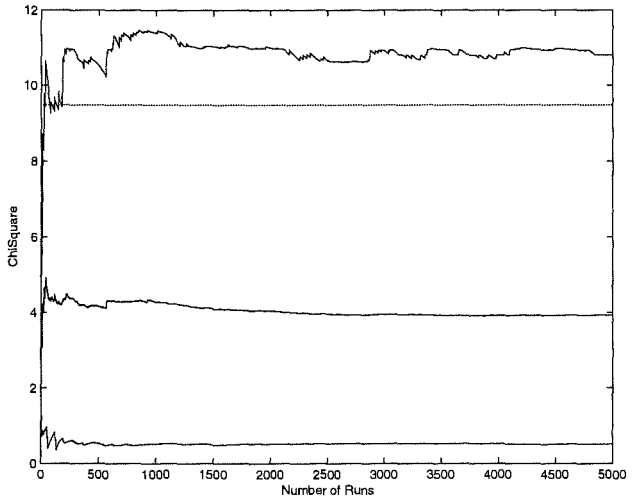


Figure 4.9: The bootstrap chi-square confidence interval as a function of the number of bootstrap samples.

The Flury test (Chapter 3) suggested that there was not a common component structure between males and females at baseline for personality at the 10% level of significance. The methods of the Flury chi-square test and the unbootstrapped multigroup analysis give the same result that there is not an underlying common structure for personality at baseline across males and females. The transformed bootstrap result gives a point estimate that is in contradiction to this, however the confidence interval is large going from a p-value of 0.03 to 0.97. So in general the methods agree that there are significant differences between males and females for the baseline personality (testing using the available female structure).

Multigroup Testing of all Models

As shown above the bootstrapping was computationally intensive and the sample size was too small to get conclusive results, evidenced by a large confidence interval for the chi-square test statistic. The other fit indices were not used as the chi-square is the most appropriate index for a strict comparison test between groups. In light of this the

Data	Model	Fit Function	χ^2	df	P-value
TCI B	Female	MLM	48.91	7	0.00
		ML	10.43	4	0.03
TCI E	Female	MLM	23.07	7	0.00
		ML	8.35	4	0.08
SCL B	Male & Female	MLM	207.25	43	0.00
		ML	219.23	40	0.00
SCL E	Female	MLM	66.56	31	0.00
		ML	108.8	26	0.00
TCISCL B	Female 7IC	MLM	179.25	32	0.00
		ML	209.34	29	0.00
	Female 10IC	MLM	602.85	60	0.00
		ML	616.50	57	0.00
	Male 6PC	MLM	213.07	53	0.00
		ML	197.00	49	0.00

Table 4.15: The multigroup analysis (hypothesis of common form) results for all models using the maximum likelihood chi-square (ML) and the mean adjusted chi-square (MLM). Key: B = Baseline, E = After Treatment.

rest of the multigroup analysis will use the maximum likelihood chi-square and the mean adjusted chi-square calculated in MPlus (Muthén and Muthén, Version 2, www.statmodel.com).

Table 4.15 presents the multigroup analysis results for all the models. The table has each of the models developed in Section 4.2. Each of these models was tested in the multigroup manner, comparing males and females. This means that where there is both a male and female model, both models have been tested for a gender effect. All the results are significant implying that for all the models there are significant differences between males and females. This matches the results from the Flury test (Chapter 3), except for the males and females baseline symptoms. The Flury test results showed there was a common structure for the males and females baseline symptoms. The multigroup analysis suggests that the males and females are significantly different on the modelled structure. This means that either the confirmatory factor analysis did not find the common structure, the sample size is too small or normality issues are playing a role. The investigation of the baseline female personality model with bootstrapping showed that the sample size was probably too small, the symptom model has more parameters to estimate from the same sample size so bootstrapping will be unable to resolve this issue.

To further investigate this problem the baseline symptom data was split into two

Group	Method	Chi-square	df	p-value
Most depressed	MLM	293.802	43	0.00
	ML	288.94	40	0.00
Least Depressed	MLM	191.32	43	0.00
	Ml	233.95	40	0.00

Table 4.16: Multigroup analysis on the most depressed and least depressed for the baseline symptom structure

groups, most depressed and least depressed. Table 4.16 presents the results. The most depressed patients have a much higher chi-square value than the least depressed patients but both have significant p-values.

One would expect with the baseline symptoms having common components (from the Flury test) and the after treatment symptoms not, that the more severely depressed the patients are more similar the symptom pattern would be across gender. However the result in Table 4.16 shows that the more depressed patients had a higher chi-square value for the multigroup analysis, suggesting more differences for the most depressed group. The only way to fully investigate these interesting quirks of the dataset is to use a much larger sample size to reduce the bootstrapped confidence interval.

4.3.2 Summary of Multigroup Analysis

The multigroup analysis method was used to investigate gender similarity and differences in the models developed from PCA, ICA, FA and then the CFA. Initial multigroup analysis in Chapter 3 used the Flury chi-square test to investigate the hypothesis of common components across gender. This test found that there were significant, or very nearly significant, gender differences across personality at both time points and symptoms after treatment. The baseline symptoms were found to have common components. This section has used multigroup analysis in the structural modelling framework to further investigate these results. The same significant differences were found for the personality models at both time points and the symptom model after treatment. The baseline symptom model was also found to be significantly different for males and females in contradiction to the Flury result. However the analysis of the bootstrap confidence interval highlighted problems with sample size for this type of analysis. This contradiction of results may right itself with a larger sample size. This would be a very interesting area for future work when a larger sample is available.

Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
FTCIB	0.698	0.428	0.008	0.344	0.469	282.208	0.462
FTCIE	0.684	0.402	0.009	0.370	0.381	330.316	0.379
FTCIBE	0.764	0.553	0.008	0.289	0.530	193.589	0.518
MTCIE	0.752	0.530	0.007	0.261	0.653	67.737	0.620
FSCLE	0.776	0.677	0.066	0.1516	0.838	193.907	0.804
FSCLBE	0.787	0.635	0.237	0.1516	0.859	185.888	0.830

Table 4.17: The results from the longitudinal models.

4.4 Longitudinal Analysis

The data contains information for the depressed patients before and after treatment (longitudinal data). This section uses structural equation modelling to model the data across time in one overall model. The latent factors are allowed to covary across time, the size and sign of the covariance gives information on the stability or change in the latent factors across time. The regression parameters are also allowed to be different across time and these can be investigated for across time effects.

For this dataset there are three different models that can be tested; the baseline model across time, the post treatment model across time and the baseline and post treatment model combined, which we would expect to perform the best. The combined TCI and SCL models have too many parameters for the number of observations when investigating in a longitudinal framework.

The results of the longitudinal tests are presented in Table 4.17. Three models were tested in a longitudinal framework for the personality of the depressed females. The first model used the baseline structure to model both time points (FTCIB), the second model uses the after treatment model for both time points (FTCIE), and the third longitudinal model uses both the baseline and after treatment model in one overall model (FTCIBE). For the personality of the depressed males, the results of the confirmatory analysis found no model with reasonable fit indices. There was however, an after treatment model found and this model is used for the longitudinal analysis (MTCIE).

The males and females were combined for the baseline symptoms and a six component model was retained. This structure is used as the first longitudinal symptom model for the females and investigates this baseline structure at both time points (FSCLB), however due to problems with non-positive definite matrices this model was not calculated. The females also had a reasonable after treatment symptom model that is investigated in the longitudinal scenario (FSCLE). The third longitudinal symptom model for the females uses the baseline and post treatment models (FSCLBE). The males did not have

a reasonable after treatment symptom model so only the baseline model is investigated in a longitudinal scenario (MSCLB). This model had problems with non-positive definite matrices so could not be calculated. Attempts to resolve this problem were unsuccessful.

The results in Table 4.17 shows that the depressed females personality is best described by the baseline and post treatment model. The baseline and post treatment model best describes the female's symptoms and the males only had the baseline model to use. As expected the combination of the best model at each time point was the best fit for modelling the data across time.

The Females Longitudinal Personality Model

Figure 4.10 presents the model for the depressed females personality across time. The best baseline and post treatment model were used to investigate the changes in personality across time. The confidence intervals for the parameters (from bootstrapping) do not include zero, so the loadings of the variables on the underlying factors are all significant. At both time points, self directedness has an error variance that is not significantly different from zero, as evidenced by the confidence interval including zero. At baseline cooperativeness is important in the model. After treatment cooperativeness is replaced by self transcendence. Cooperativeness is an important personality trait for the females when they are depressed (at baseline) and self transcendence is important after treatment. Harm avoidance, persistence and self directedness are also important in describing the female's personality at both time points. The loadings for these three variables remain similar across time (Figure 4.10). There is a significant covariance across time, with the confidence interval totally positive (Figure 4.10). This suggests that those who are high in harm avoidance, and low in persistence, self directedness and cooperativeness at baseline will be high in harm avoidance and low in persistence, self directedness and self transcendence after treatment.

Table 4.18 presents the fit indices (bootstrapped) for the female's longitudinal personality model. Most of the fit indices are in the appropriate bounds however some of the confidence intervals are just outside the ideal bounds. For example the lower bound on the CFI is 0.944 and ideally the CFI should be above 0.95. The chi-square value, as expected has a large interval and is inconclusive suggesting that for this strict measure of fit a larger sample is needed. However, given that the other fit indices appear well behaved the model may be appropriate.

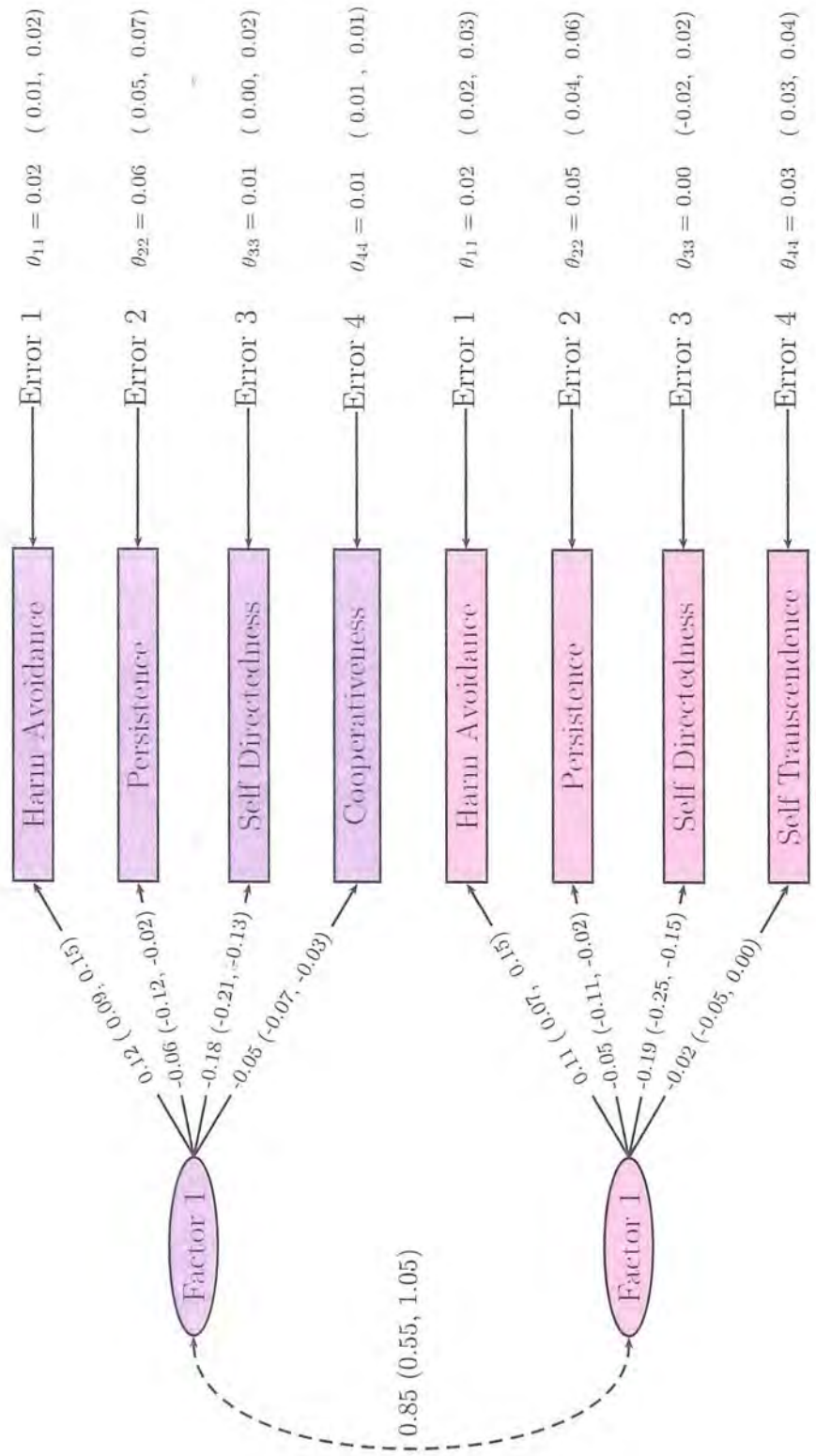


Figure 4.10: The females longitudinal TCI model.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.751	(0.710, 0.787)	0.966	(0.943, 0.982)
AGFI	0.528	(0.450, 0.597)	0.935	(0.891, 0.965)
RMR	0.008	(0.006, 0.010)	0.002	(0.001, 0.003)
Chi-Square	248.299	(197.694, 305.834)	19.551	(10.201, 33.639)
df	19		19	
p-value	0.000	(0.000, 0.000)	0.422	(0.948, 0.020)
RMSEA	0.300	(0.265, 0.336)	0.015	(0.000, 0.076)
CFI	0.523	(0.441, 0.601)	0.998	(0.944, 1.000)
AIC	210.299	(159.694, 267.834)	-18.449	(-27.799, -4.361)
NFI	0.512	(0.434, 0.586)	0.929	(0.880, 0.963)

Table 4.18: The bootstrapped fit indices for the females longitudinal TCI model.

Figure 4.11 presents boxplots for the estimated factor scores before and after treatment. The boxplots show that the factor scores have a similar distribution over time. This suggests that the structural equation modelling has found a stable personality factor across time, the difference between the time points is the replacement of cooperativeness by self transcendence.

The Females Longitudinal Symptom Model

Figure 4.13 presents the female's longitudinal model. The baseline part of the model consists of the six factor model found using the confirmatory factor analysis approach and the after treatment model is the two factor model found to best fit the post treatment symptoms.

Table 4.19 presents the covariances between the latent factors of Figure 4.13. The confidence intervals are non-zero and positive indicating all covariances are significant and all are positively related. Thus the factors are highly related across time. This suggests that a person comparatively high in symptoms at baseline will be comparatively high in symptoms post treatment. This does not necessarily mean that the symptoms have not improved, just that compared to the other people in the group the high symptom group are likely to be the high symptom group after treatment.

Table 4.20 presents the fit indices for the model. The transformed indices show the model has a reasonable fit, though the AGFI, RMR and NFI confidence intervals do include the boundary. As expected the chi-square interval is large with an ambiguous p-value confidence interval. However taking into account all the fit indices and in view of

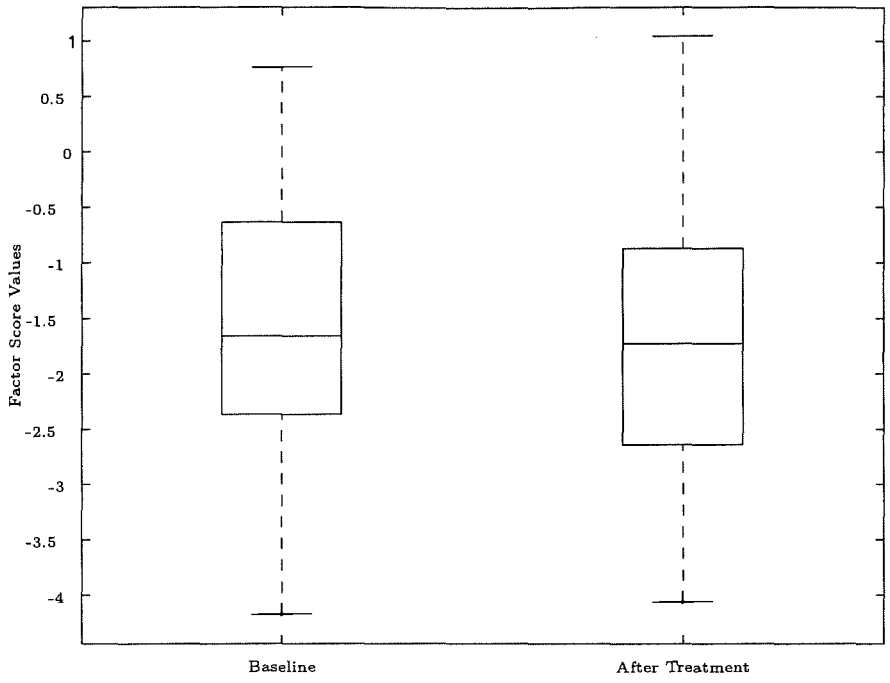


Figure 4.11: Boxplots of the female TCI factor scores at baseline and after treatment.

Covariance	Median	(90% CI)	Covariance	Median	(90% CI)
Factor 1 Factor 2	0.881	(0.768, 0.942)	Factor 1 Factor 3	0.900	(0.864, 0.930)
Factor 2 Factor 3	0.841	(0.797, 0.880)	Factor 1 Factor 4	0.819	(0.745, 0.875)
Factor 2 Factor 4	0.871	(0.818, 0.913)	Factor 3 Factor 4	0.722	(0.666, 0.771)
Factor 1 Factor 5	0.852	(0.809, 0.889)	Factor 2 Factor 5	0.738	(0.670, 0.795)
Factor 3 Factor 5	0.763	(0.716, 0.805)	Factor 4 Factor 5	0.725	(0.663, 0.777)
Factor 1 Factor 6	0.584	(0.428, 0.700)	Factor 2 Factor 6	0.659	(0.519, 0.758)
Factor 3 Factor 6	0.553	(0.426, 0.645)	Factor 4 Factor 6	0.449	(0.247, 0.606)
Factor 5 Factor 6	0.441	(0.293, 0.557)	Factor 1 Factor 7	0.449	(0.257, 0.612)
Factor 2 Factor 7	0.432	(0.246, 0.597)	Factor 3 Factor 7	0.433	(0.272, 0.569)
Factor 4 Factor 7	0.406	(0.218, 0.585)	Factor 5 Factor 7	0.501	(0.326, 0.639)
Factor 6 Factor 7	0.336	(0.128, 0.505)	Factor 1 Factor 8	0.446	(0.274, 0.602)
Factor 2 Factor 8	0.557	(0.379, 0.707)	Factor 3 Factor 8	0.488	(0.328, 0.612)
Factor 4 Factor 8	0.474	(0.286, 0.645)	Factor 5 Factor 8	0.439	(0.270, 0.580)
Factor 6 Factor 8	0.339	(0.168, 0.493)	Factor 7 Factor 8	0.956	(0.910, 0.987)

Table 4.19: The bootstrapped parameter estimates for the females longitudinal SCL model.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.744	(0.709, 0.775)	0.926	(0.897, 0.948)
AGFI	0.586	(0.529, 0.636)	0.880	(0.833, 0.916)
RMR	0.226	(0.179, 0.277)	0.044	(0.022, 0.102)
Chi-Square	463.466	(393.408, 543.691)	108.406	(72.425, 158.820)
df	84		84	
p-value	0.000	(0.000, 0.000)	0.038	(0.812, 0.000)
RMSEA	0.169	(0.152, 0.186)	0.043	(0.000, 0.075)
CFI	0.818	(0.780, 0.851)	0.990	(0.970, 1.000)
AIC	295.462	(225.408, 375.691)	-59.603	(-95.603, -9.192)
NFI	0.789	(0.753, 0.822)	0.958	(0.938, 0.971)

Table 4.20: Bootstrapped Fit Indices for the Females Longitudinal SCL Model

the sample size available the model is a reasonable representative of the female symptoms across time.

Figure 4.12 presents the distribution of the factor scores for the female's symptoms. At baseline there are 6 factors, the last four are single indicator variables. After treatment there are 2 factors. The factor scores after treatment are smaller, on average, than most of the baseline factor scores. The distributions are right skewed after treatment. The factor loadings are smaller after treatment (Figure 4.13), showing that the depressed patients have improved.

The Males Longitudinal Personality Model

Figure 4.15 presents the male's longitudinal personality model. During the confirmatory analysis no baseline model was found that was sufficiently well fitting. The post treatment has been used to model both the baseline and post treatment symptoms because of this. At both time points harm avoidance has a significant positive loading and the other three variables, persistence, self directedness and cooperativeness have significant negative loadings. There is a significant covariance across time.

The fit indices are presented in Table 4.21. Most of the fit indices have confidence intervals that are ambiguous. For example the CFI has a lower bound of 0.913, well below the 0.95 criteria for good fit. A definitive answer on the fit of this model would need a larger sample size. The fit indices are in the right bounds but the confidence intervals are large.

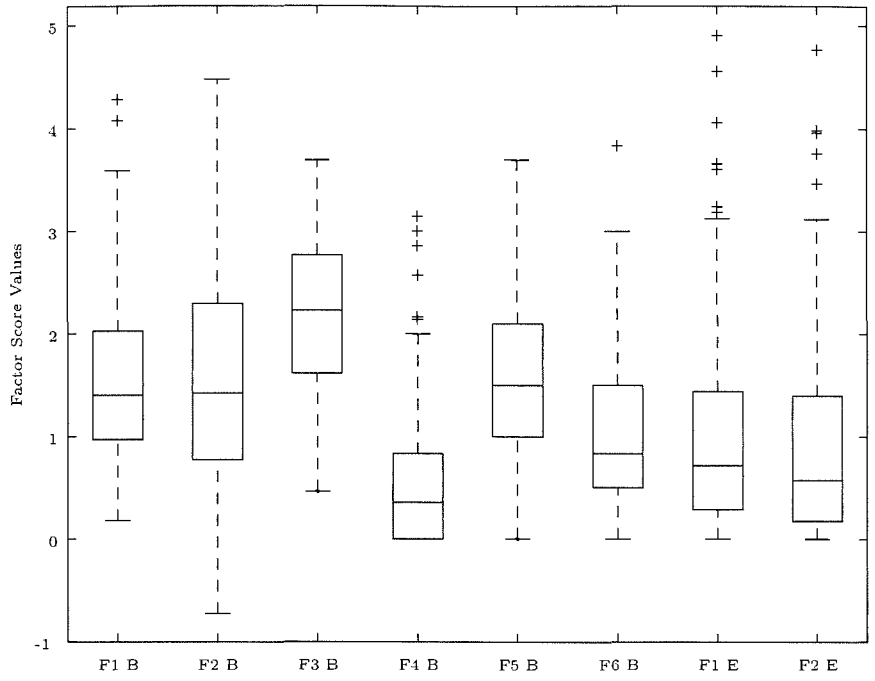


Figure 4.12: Boxplots of the female SCL factor scores at baseline and after treatment. Key: B = Baseline; E = After Treatment.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.726	(0.669, 0.777)	0.937	(0.893, 0.965)
AGFI	0.482	(0.372, 0.578)	0.881	(0.797, 0.934)
RMR	0.007	(0.005, 0.008)	0.002	(0.002, 0.003)
Chi-Square	124.537	(96.558, 160.229)	19.058	(9.926, 34.873)
df	19		19	
p-value	0.000	(0.000, 0.000)	0.453	(0.955, 0.014)
RMSEA	0.288	(0.247, 0.333)	0.007	(0.000, 0.112)
CFI	0.621	(0.506, 0.716)	1.000	(0.913, 1.000)
AIC	86.537	(58.558, 122.229)	-18.942	(-28.074, -3.127)
NFI	0.593	(0.489, 0.683)	0.904	(0.828, 0.949)

Table 4.21: The bootstrapped fit indices for the males longitudinal TCI model.

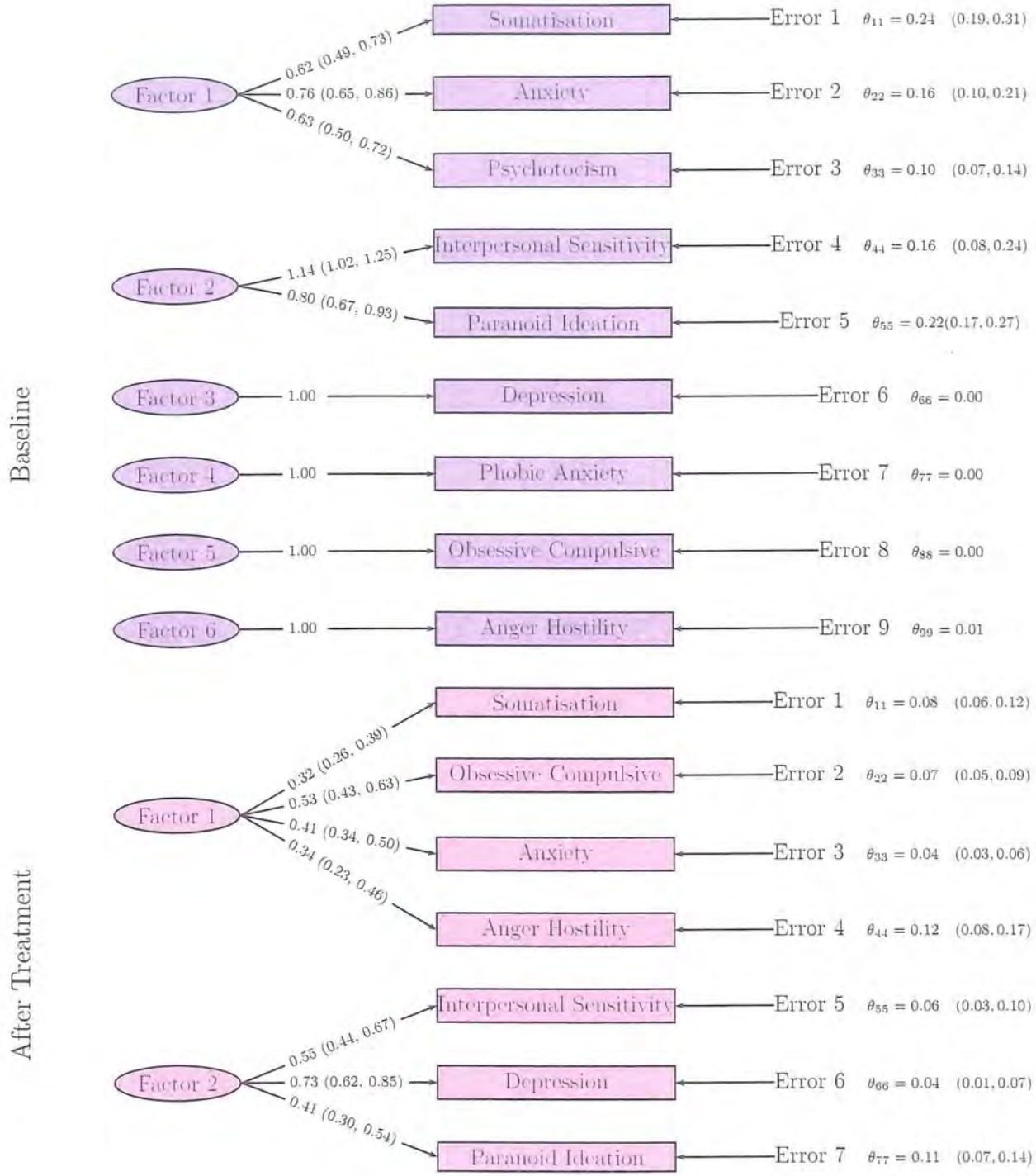


Figure 4.13: Depressed Females Longitudinal Symptom Model

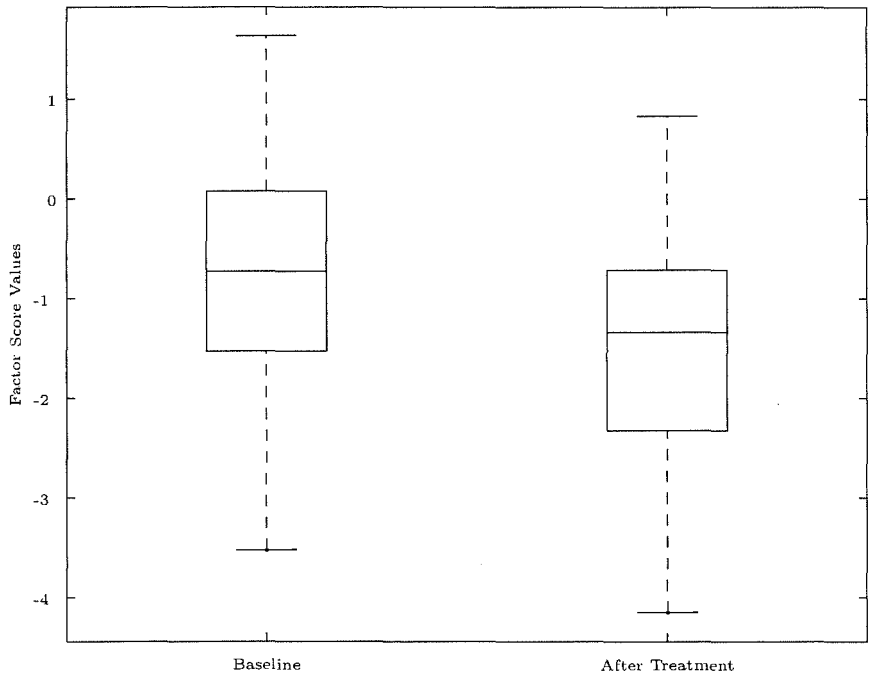


Figure 4.14: Boxplots of the male TCI factor scores at baseline and after treatment.

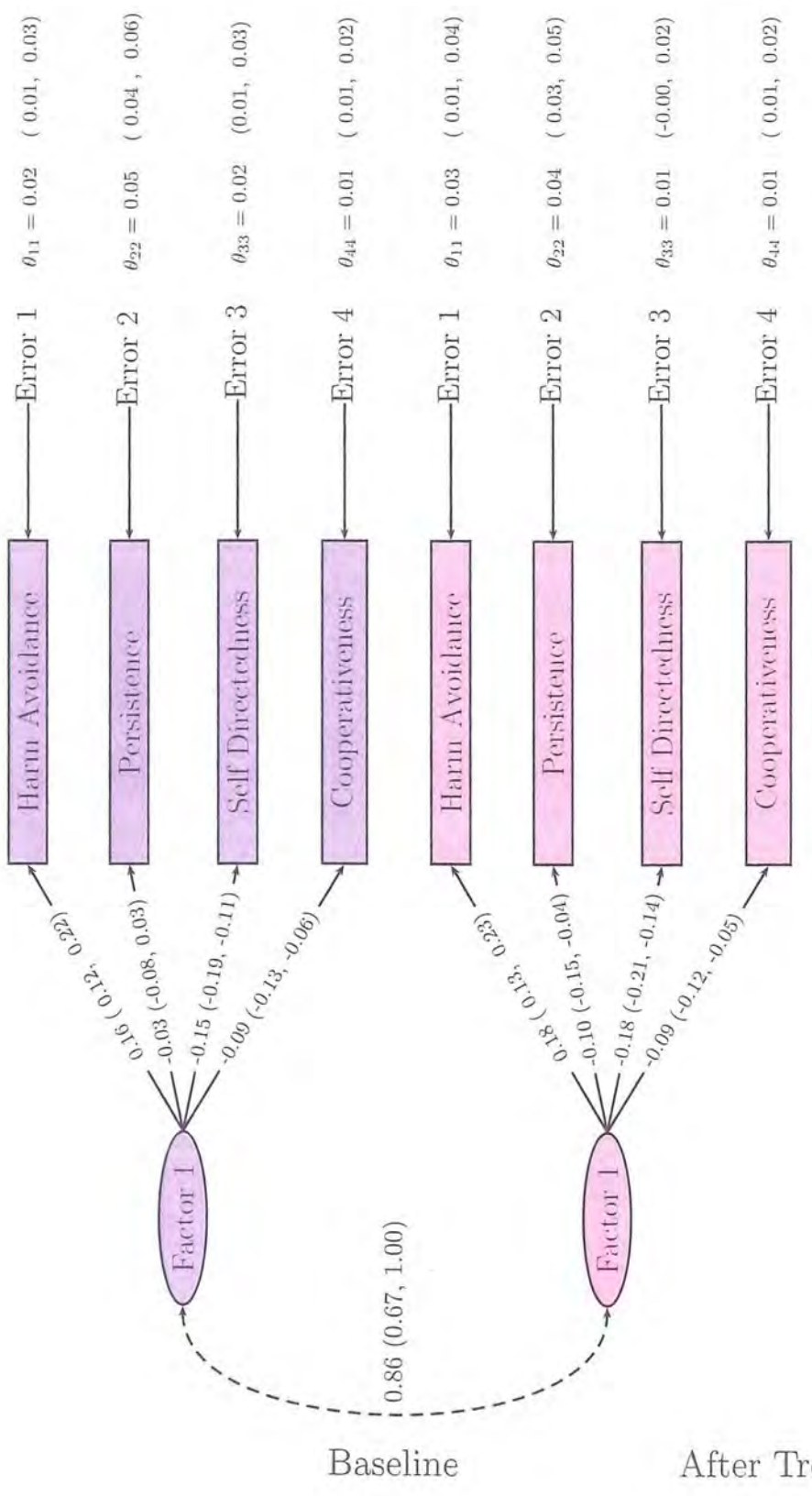


Figure 4.15: The males longitudinal TCI model.

Figure 4.14 presents boxplots for the estimated factor score distribution for the males personality before and after treatment. The graph shows that factor scores are smaller after treatment. The factor loadings are similar across time (Figure 4.15) so this suggests that over time the combination of harm avoidance versus persistence, self directedness and cooperativeness has decreased. So either harm avoidance has decreased or persistence, self directedness or cooperativeness have increased or a combination of both has happened.

4.5 SEM Factors as Predictors and Discriminators of Depression Outcome

Chapter 4 has presented the best components to describe the covariance structure of the personality and symptom data. This section presents an investigation into the use of these components as discriminators between high and low depression scores on the Hamilton Depression Rating. Discriminant analysis was conducted on the Hamilton scores categorised using quartiles. The idea being, would the baseline symptom and personality factors significantly discriminate depression outcome measured by the Hamilton score.

4.5.1 Discriminant Analysis

Discriminant analysis is used to classify data into predetermined groups. The functions that are used to do this can then be used to predict groupings for new observations. The theory presented here is from the SAS/STAT User's Guide (1999, SAS Institute Inc., USA) and is reproduced here for convenience. We are following this theory as it better represents the options chosen for the discriminant analysis conducted in SAS (SAS(R) Proprietary Software Release (8.1)). Further details for discriminant analysis can be found in Morrison (1976).

Discriminant analysis groups the data by finding the grouping that the observation is closest to. Distance is measured by the Mahalanobis distance which measures how far observations ($\mathbf{x}$) are away from group i. The squared distance is

$$D_i^2(\mathbf{x}) = (\mathbf{x} - \mathbf{m}_i)'\mathbf{V}_i^{-1}(\mathbf{x} - \mathbf{m}_i), \tag{4.47}$$

where $\mathbf{m}_i$ is the vector containing the group i variable means and V_i is the pooled covariance matrix. The density estimate at $\mathbf{x}$, for group i with p variables, is the function

$$f_i(\mathbf{x}) = (2\pi)^{-\frac{p}{2}}|\mathbf{V}_i|^{-\frac{1}{2}}e^{-0.5D_i^2(\mathbf{x})}. \tag{4.48}$$

The posterior probability of $\mathbf{x}$ belonging to group i can be calculated using Bayes' theorem

$$p(i|\mathbf{x}) = \frac{q_i f_i(\mathbf{x})}{\Sigma_u q_u f_u(\mathbf{x})} \tag{4.49}$$

$$\tag{4.50}$$

$$= \frac{e^{-0.5D_i^2(\mathbf{x})}}{\Sigma_u e^{-0.5D_u^2(\mathbf{x})}}, \tag{4.51}$$

for q_i, the prior probability of belonging to group i.

The grouping is chosen through the use of the discriminant scores, $-0.5D_u^2(\mathbf{x})$. Classification is achieved by setting $i = u$, if this produces the largest $p(i|\mathbf{x})$ or the smallest value of $D_i^2(\mathbf{x})$ then the observation is classified into group u.

Stepwise Discriminant Analysis

SAS PROC STEPDISC (SAS(R) Proprietary Software Release (8.1)) was used to perform stepwise discriminant analysis. This routine adds and removes variables to achieve the maximum group separation. The tests were performed at the 10% significance level and the routine stops when no further variables can be added or removed.

Test for the Equality of the Covariance Matrices

All the models use the pooled covariance matrix. Before using the pooled matrix the equality of the covariance matrices was tested using the Chi-square test from Morrison (1976) with a null hypothesis of

$$H_0 : \boldsymbol{\Sigma_1} = \cdots = \boldsymbol{\Sigma_k}$$

and a test statistic of

$$M = \sum n_i \ln |\mathbf{S}| - \sum_{i=1}^{k} n_i \ln \mathbf{S}_i \tag{4.52}$$

where $\mathbf{S}$ is the pooled covariance matrix. When multiplied by a specific scale factor the distribution is approximately chi-squared and this approximation is good if k and p do not exceed about five (Morrison, 1976). All the models presented below were found to have no significant differences between covariance matrices and thus the pooled covariance matrix is used in the analyses.

Sensitivity and Specificity

Taube (1986) presents some graphical analyses that can be made from the cross-classification table of the actual groupings and the predicted groupings from the discriminant analysis. The following conditional probabilities are defined

$$P(\text{'Predicted 1'}|\text{'Actual 1'}) = \text{'sensitivity'} = r \tag{4.53}$$
$$P(\text{'Predicted 0'}|\text{'Actual 0'}) = \text{'specificity'} = s. \tag{4.54}$$

Also, the prevalence of the actual ones (group A1) is denoted as P. The following notation will be used.

- True Positives (TP) are those who are predicted to be 1 and were 1.
- False Positives (FP) are those who are predicted to be 1 but were actually 0.
- True Negatives (TN) are those who are predicted to be 0 and were 0.
- False Negatives (FN) are those who are predicted to be 0 but were actually 1.

When the prediction is unrelated to the actual result, $r + s = 1$ thus one measure of test efficiency is the Youden index (Youden, 1950) $J = r + s - 1$. In one random sample of size n, where there is independent information about the actual 0 or 1 classification, the sensitivity and specificty can be estimated as

$$\widehat{r} = TP/(TP + FN) \tag{4.55}$$
$$\widehat{s} = TN/(FP + TN) \tag{4.56}$$
$$\widehat{P} = (TP + FN)/n \tag{4.57}$$

The index of validity can also be defined as the proportion of correctly classified individuals ($I_v = (TP + TN)/n$). This index of validity can be rewritten in terms of the sensitivity and specificity as (Feinstein, 1977)

$$I_v = (r - s)P + s. \tag{4.58}$$

In the situation of the sensitivity and specificity being equal, the first term of the equation is zero and the index of validity is independent of the prevalence.

Taube (1986) presents a graphical picture of these concepts. The graph has an x-axis that represents the prevalence and the y-axis is the relative frequencies of the observations. The line $y = rx$ indicates the sensitivity and $y = sx + (1 - s)$ indicates the specificity. The area between the sensitivity and specificity lines represent the false negatives and false positives. The vertical line $x = P$ represents the expected frequencies in the cross classification table. This graphical technique will be used in the next section to aid interpretation of the results.

4.5.2 Results from the Discriminant Analysis

The components developed in the structural equation modelling will be used as the predictors of Hamilton Score to see if these new components can significantly discriminate levels of the Hamilton Score. The Hamilton score has been split using the lower quartile, median and upper quartile. Three classifications are investigated, firstly those observations above the upper quartile versus the rest (G4 vs G3 G2 G1); those observations above the median versus those below (G4 G3 vs G2 G1); and those observations above the lower quartile versus those below (G4 G3 G2 vs G1).

As would be expected the baseline symptom and personality components significantly discriminate the baseline level of the Hamilton Score (Table 4.22). This is expected as more severe depression will cause more severe symptoms. Interestingly personality also is involved in some of the discriminating functions, and this possibly due to the fact that harm avoidance and self directedness particularly, tend to change with depression severity.

What we are really interested however, is the depression outcome. Do the baseline components offer a better insight into the post treatment Hamilton score and the change in Hamilton score (post treatment Hamilton minus baseline Hamilton). Table 4.23 presents the variables that significantly discriminate post treatment Hamilton scores. The second and third symptom components significantly discriminate the lowest Hamilton score levels (less than the lower quartile). For the males the personality component significantly discriminates between Hamilton scores below the median and above the median. With the exception of those two models the components are poor discriminators of post treatment Hamilton score. Investigation of the Hamilton score after treatment however does not take into account how severe the depression was to begin with. So the change in Hamilton score (post treatment minus baseline) was investigated.

Table 4.24 presents the component or component combinations that significantly discriminate levels of the change in Hamilton score. There are more models for the different quartile level comparisons. Change in Hamilton score is measured by post treatment minus baseline. The more the symptoms improve the more negative the change in symptoms. Thus having a cutpoint of the upper quartile is comparing the 25 percent with the least improvement to the rest of the observations. The models that significantly discriminate the change in Hamilton score will be further analysed in the next section.

Discriminant Analysis for the Depressed Females Change in Hamilton Score using the Lower Quartile as the Cutpoint

One model significantly discriminated the change in Hamilton score for the depressed females. The cut point was chosen as the lower quartile for the change in Hamilton score. This groups the least improved 75% (coded as 1) against the most improved 25% (coded

	Model	Components that Discriminant
Female	G4 vs G3 G2 G1	F1SCL F4SCL F5SCL F3SCL
	G4 G3 vs G2 G1	F1SCL F2SCL F1TCI
	G4 G3 G2 vs G1	F1TCI F1SCL F2SCL
Male	G4 vs G3 G2 G1	F3SCL F4SCL
	G4 G3 vs G2 G1	F1SCL F2SCL F3SCL F5SCL
	G4 G3 G2 vs G1	F1SCL F2SCL

Table 4.22: Baseline components that significantly discriminant baseline Hamilton scores.

	Model	Components that Discriminant
Female	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	None
	G4 G3 G2 vs G1	F3SCL F2SCL
Male	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	None
	G4 G3 G2 vs G1	None

Table 4.23: Baseline components that significantly discriminant post treatment Hamilton scores.

	Model	Components that Discriminant
Female	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	None
	G4 G3 G2 vs G1	F1SCL
Male	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	F1SCL F2SCL
	G4 G3 G2 vs G1	F1SCL F2SCL

Table 4.24: Baseline components that significantly discriminant change in Hamilton scores (post treatment minus baseline).

Variable	0	1
Constant	-3.25054	-2.20820
F1SCL	1.36977	1.12899

Table 4.25: The linear discriminant function for the depressed females change in Hamilton score, using the lower quartile as the cut point.

	True Change in Hamilton score		
	1	0	Total
1	30	9	39
classified			
0	18	11	29
Total	48	20	68

Table 4.26: The number of observations classified into change in Hamilton score for the depressed females, using the lower quartile as the cut point.

as 0). The first symptom component (F1 SCL) was found to be a significant discriminator. Wilks' Lambda for this model is 0.96 and has a p-value of 0.0969, so there is significant discrimination at the 10% level of significance.

The linear discriminant function is presented in Table 4.25. Table 4.26 presents the cross classification with the columns showing the true classification and the rows presenting the classification by the discriminant function. From the table the sensitivity and specificity are 0.63 and 0.55 respectively, with a prevalence of poor improvement in the Hamilton score of 0.71. This number is different from the 0.75 expected from the quartile grouping. This is caused by observations that are equal to the lower quartile. The grouping was classified as strictly greater than the particular quartile level rather than a fifty fifty split of observations on the quartile level. If a fifty fifty split was chosen, one could only randomly assign the grouping of zero or one. It is better to have a set cut point and a predefined include or exclude rule as used in this analysis. However this does lead to the confusing prevalence rate.

Figure 4.16 presents the normal density estimates from the discriminant analysis. The density curves suggest that higher F1 SCL values predict the most improvement in the Hamilton Score (DY=0). Likewise the posterior probabilities (Figure 4.17) show that high values of F1 SCL are predictive of the most improved group (the bottom 25% of the change in Hamilton score).

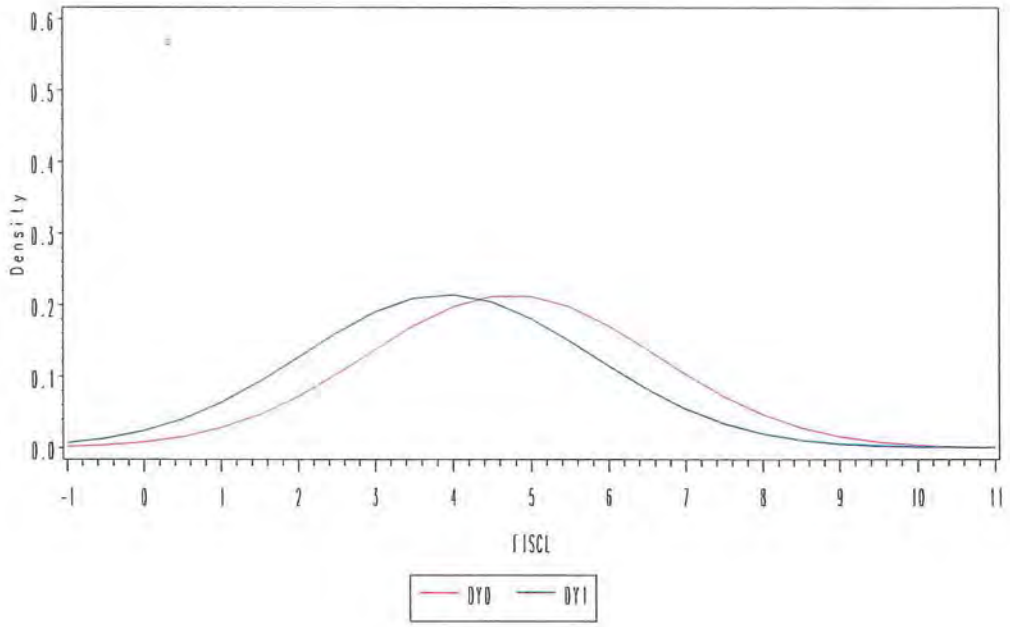


Figure 4.16: The normal density estimates for the depressed females change in Hamilton score, using the lower quartile as the cut point.

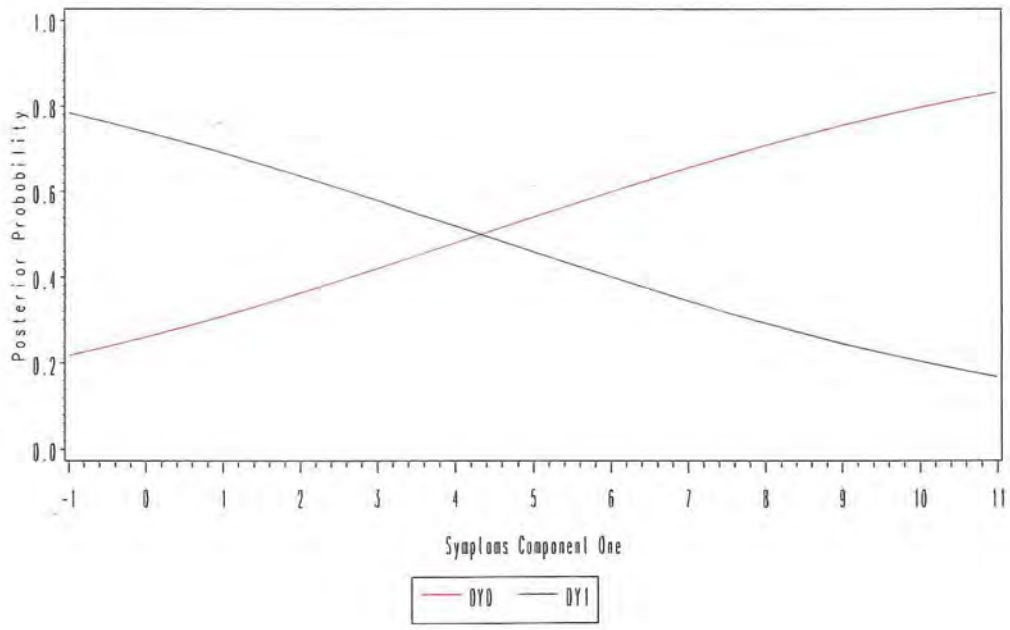


Figure 4.17: The posterior probabilities for the depressed females change in Hamilton score. using the lower quartile as the cut point.

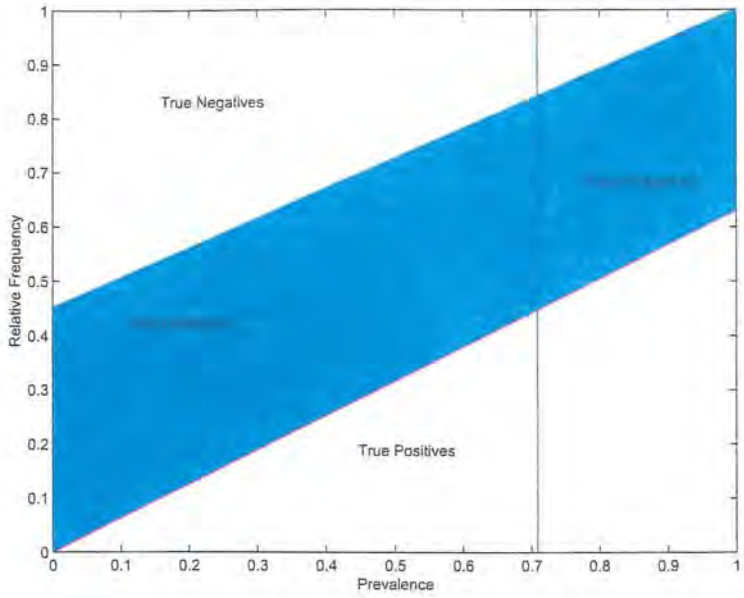


Figure 4.18: Sensitivity and specificity for varying prevalence levels for the depressed females change in Hamilton score, using the lower quartile as the cut point.

Figure 4.18 shows how the sensitivity and specificity change with differing prevalences. On the graph the bottom line represents the sensitivity and the top line the specificity. The graph represents the expected frequencies along a given prevalence line. The shaded area represents the false positives and false negatives. For this prevalence the sensitivity is greater than the specificity. Also the number of false negatives is larger than the number of false positives. A false negative occurs when a person is classified as a good improver when in fact they were a poor improver. Conversly a false positive is when a person is classified as a poor improver when in fact they were not. For the clinician one of these errors may be worse than the other because of treatment regime or some other reason. The cutpoint chosen for the change in Hamilton score can be adjusted thus changing the prevalence in a direction that reduces the unwanted error.

Discriminant Analysis for the Depressed Males Change in Hamilton Score using the Lower Quartile as the Cutpoint

The first and second symptom components significantly discriminate Hamilton scores above the upper quartile from the other scores (Wilks' Lambda $= 0.87$, $p = 0.0511$). The observations above the upper quartile are the observations with the least improvement.

The linear discriminant function is presented in Table 4.27. Table 4.28 presents the cross-classification results. The sensitivity is 0.68 and the specificity is 0.75 with a prevalence of 0.74.

Variable	0	1
Constant	-4.72370	-3.43185
F1SCL	1.78219	0.86306
F2SCL	-0.13048	0.57793

Table 4.27: The linear discriminant function for the depressed males change in Hamilton score, using the lower quartile as the cut point.

		True Change in Hamilton score		
		1	0	Total
classified	1	23	3	26
	0	11	9	20
Total		34	12	46

Table 4.28: The number of observations classified into change in Hamilton score for the depressed males, using the lower quartile as the cut point.

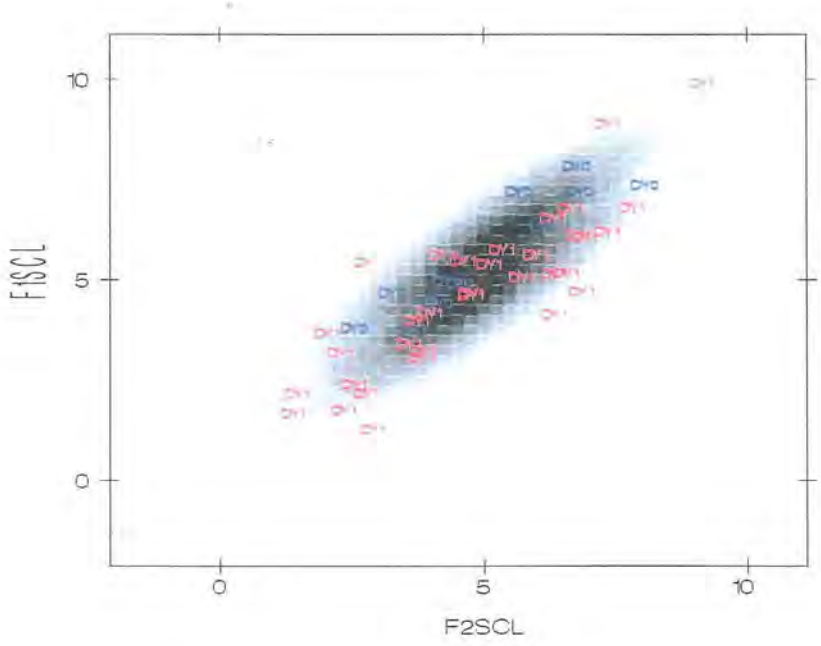


Figure 4.19: The normal density estimates for the depressed males change in Hamilton score, using the lower quartile as the cut point.

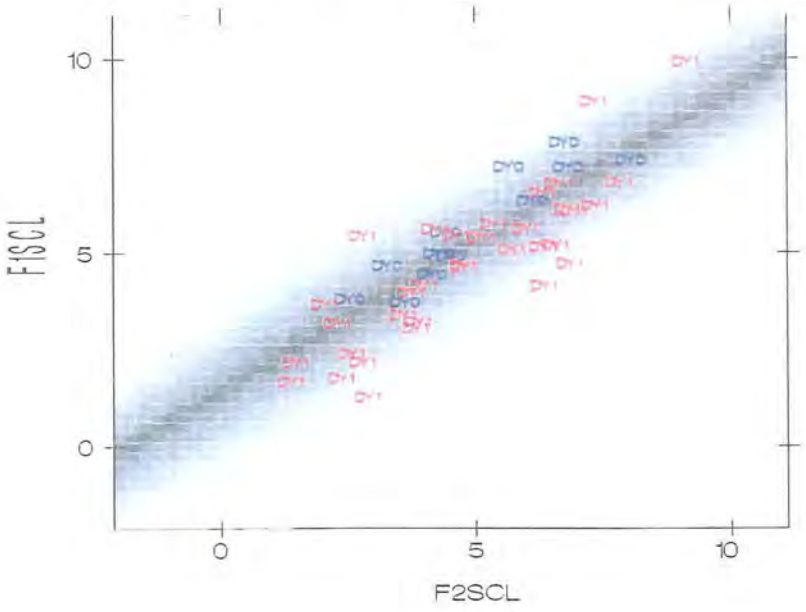


Figure 4.20: The posterior probabilities for the depressed males change in Hamilton score, using the lower quartile as the cut point (black to white is low to high probability).

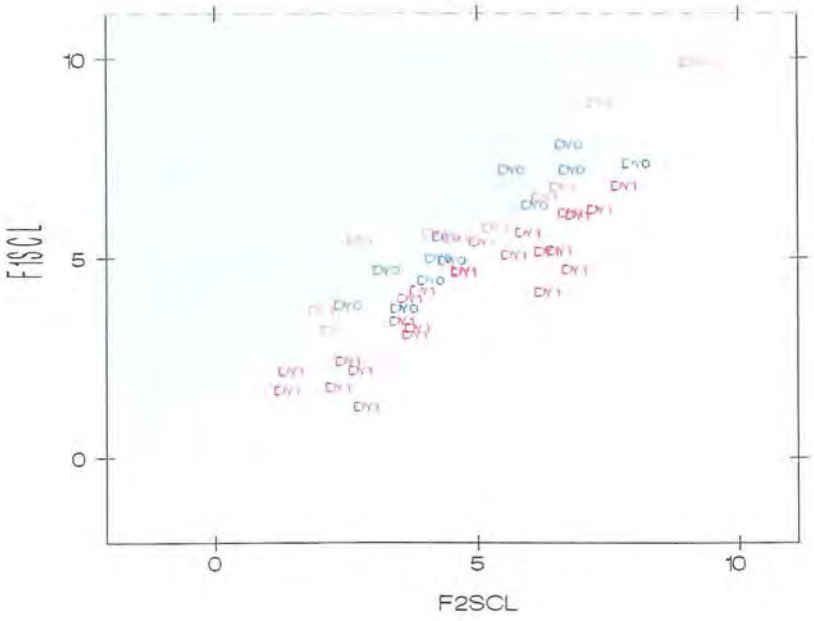


Figure 4.21: The classification results for the depressed males change in Hamilton score, using the lower quartile as the cut point.

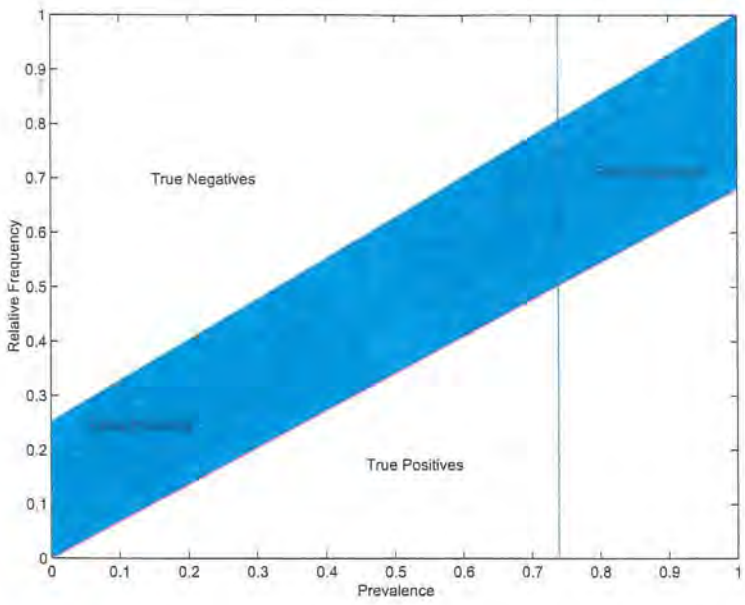


Figure 4.22: Sensitivity and specificity for varying prevalence levels for the depressed males change in Hamilton score, using the lower quartile as the cut point.

Figure 4.19 shows the normal density estimates, Figure 4.20 shows the posterior probabilities and Figure 4.21 presents the classification results. These graphs show that high values of F1SCL are associated with more improvement and low values of F2SCL are associated with more improvement. The results for F1SCL match that seen for the females with lower quartile cutpoint. Figure 4.22 presents the sensitivity and specificity results for varying prevalence levels. For the actual prevalence level there are more false negatives than false positives.

Discriminant Analysis for the Depressed Males Change in Hamilton Score using the Median as the Cutpoint

The same components, F1SCL and F2SCL, also significantly discriminate the change in Hamilton score when the median is used as the cut point (Wilks' Lambda=0.72, $p = 0.0008$). The linear discriminant function is presented in Table 4.29. Table 4.30 presents the cross-classification results. The sensitivity is 0.77 and the specificity 0.67 with a prevalence of 0.48. The sensitivity and specificity are plotted for varying prevalence levels in Figure 4.26. The graph is similar to that presented for the lower quartile cutpoint as the underlying model is similar.

The normal density estimates are plotted in Figure 4.23, the posterior probabilities are plotted in Figure 4.24 and the classification results are shown in Figure 4.25. These plots again show that high values of F1SCL are suggestive of the greater improvement and

Variable	0	1
Constant	-5.28834	-3.09593
F1SCL	2.17977	0.77628
F2SCL	-0.34115	0.63365

Table 4.29: The linear discriminant function for the depressed males change in Hamilton score, using the median as the cut point.

		True Change in Hamilton score		
		1	0	Total
classified	1	17	8	25
	0	5	16	21
Total		22	24	46

Table 4.30: The number of observations classified into change in Hamilton score for the depressed males, using the median as the cut point.

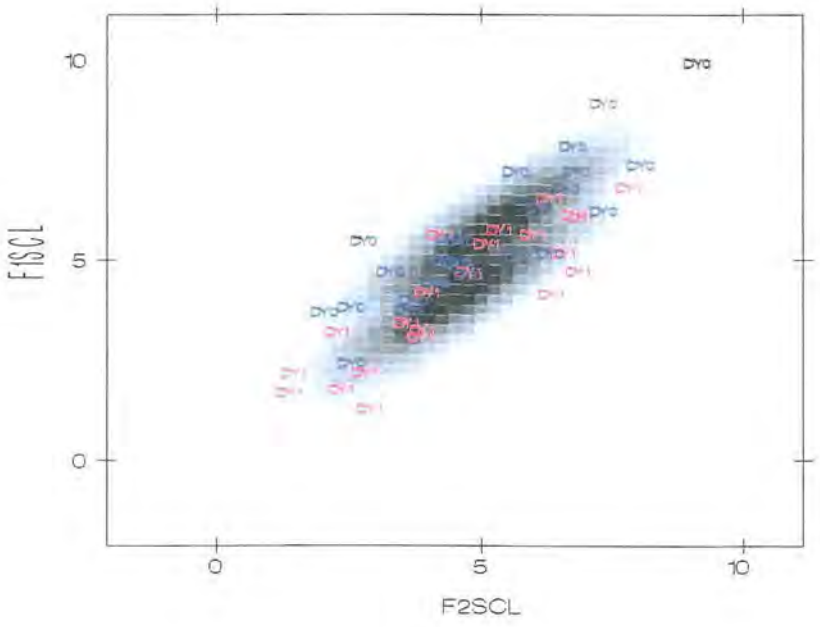


Figure 4.23: The normal density estimates for the depressed males change in Hamilton score, using the median as the cut point.

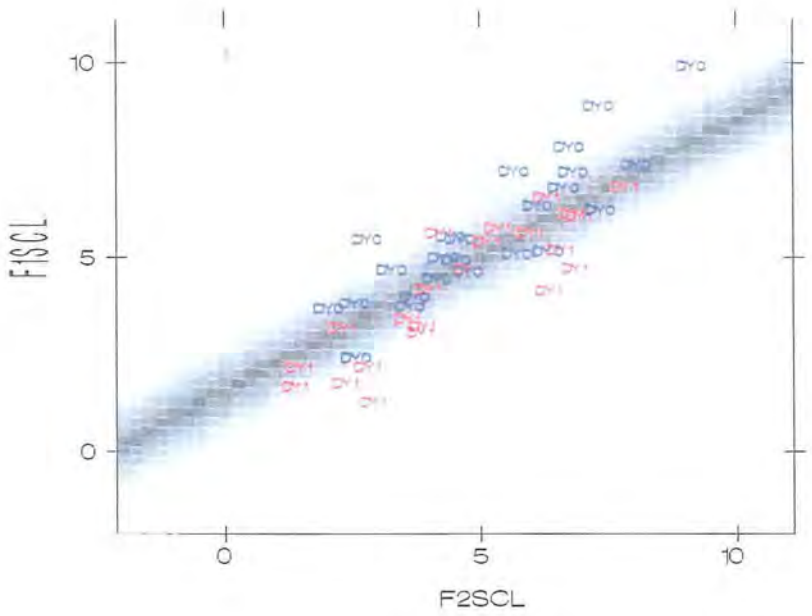


Figure 4.24: The posterior probabilities for the depressed males change in Hamilton score, using the median as the cut point (black to white is low to high probability).

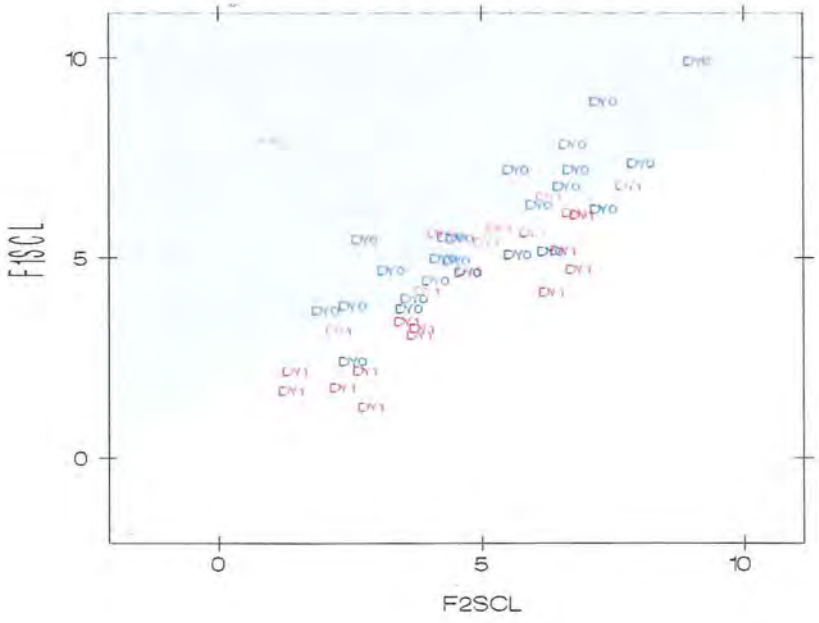


Figure 4.25: The classification results for the depressed males change in Hamilton score, using the median as the cut point.

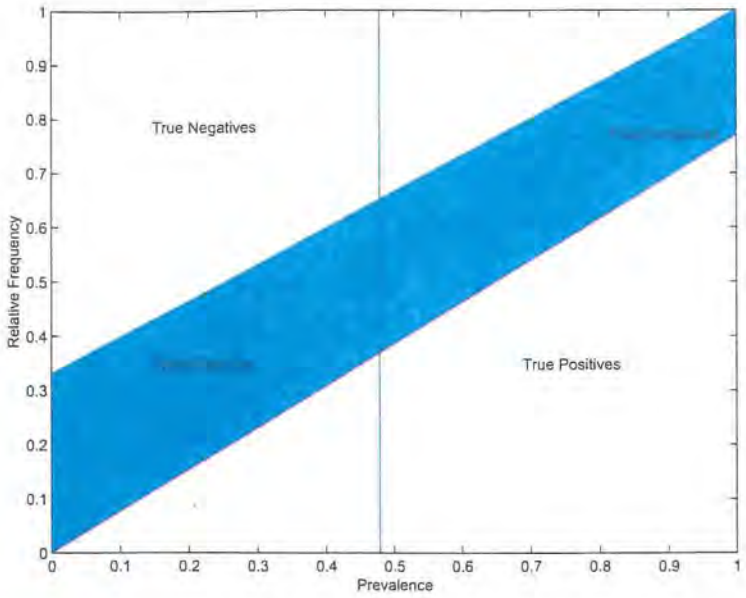


Figure 4.26: Sensitivity and specificity for varying prevalence levels for the depressed males change in Hamilton score, using the median as the cut point.

low values of F2SCL are suggestive of greater inprovement. This matches with the result presented for the males using the upper quartile cutpoint. So the change in cutpoint has not altered the model as such though the sensitivity and specificity values are different as the prevalence has changed.

4.5.3 Logistic Regression

The classifications for the change in Hamilton score used in the discriminant analysis were also used in a Logistic Regression. The components developed in this chapter were used as predictors of the binary outcome change in Hamilton score where the cut points were chosen as the quartiles.

Logistic regression is used to model a binary or dichotomous outcome variable. For this chapter the binary outcome is the change in Hamilton score using a quartile cut point q_j as follows

$$y_i = \begin{cases} 1, & \text{if } y_i > q_j \\ 0, & \text{otherwise.} \end{cases} \tag{4.59}$$

The regression model for $\mathbf{x}$, the vector of independent variables, is (Hosmer and Lemeshow, 2000)

$$g(\mathbf{x}) = ln[\frac{\pi(\mathbf{x})}{1-\pi(\mathbf{x})}] \tag{4.60}$$

$$= \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \cdots + \beta_p x_p + \epsilon, \tag{4.61}$$

where

$$\pi(x) = E(y|\mathbf{x}) = \frac{e^{g(\mathbf{x})}}{1+e^{g(\mathbf{x})}} \tag{4.62}$$

and β_p are the regression coefficients. The transformation from $\pi(\mathbf{x})$ to $g(\mathbf{x})$ is the logit transformation which means $g(\mathbf{x})$ has the desirable properties of the linear regression model. However, the binomial distribution, rather than the normal distribution in standard regression, describes the distribution of the errors (Hosmer and Lemeshow, 2000)

$$\epsilon \approx B(0, \pi(\mathbf{x})[1-\pi(\mathbf{x})]). \tag{4.63}$$

The model is fitted using maximum likelihood estimation and in practice the log likelihood is maximised to obtain the estimates of $\boldsymbol{\beta}$. The log likelihood function is (Hosmer and Lemeshow, 2000)

$$L(\boldsymbol{\beta}) = ln(\prod_{i=1}^{n} \pi(x_i)^{y_i}[1-\pi(x_i)]^{1-y_i}). \tag{4.64}$$

This function is a maximum when its first derivative is zero. This leads to the likelihood equations

$$\sum_{i=1}^{n}[y_i - \pi(x_i)] = 0 \tag{4.65}$$

$$\sum_{i=1}^{n} x_{ij}[y_i - \pi(x_{ij})] = 0 \tag{4.66}$$

for $j = 1, \ldots, p$. These equations are solved iteratively within the chosen software package. For this study SAS (SAS(R) Proprietary Software Release (8.1)) was used for the logistic regression.

Testing the Model

The first test for overall model fit is the likelihood ratio test (D), defined as (Hosmer and Lemeshow, 2000)

$$D = -2\sum_{i=1}^{n}\left[y_i\, ln\left(\frac{\widehat{\pi}_i}{y_i}\right) + (1-y_i)\, ln\left(\frac{1-\widehat{\pi}_i}{1-y_i}\right)\right]. \tag{4.67}$$

This test statistic measures the ratio of the likelihood of the model of interest to the likelihood of the saturated model (a model containing the same number of parameters and data points).

The second test for model fit used in this study is the Wald test. For the data matrix $\mathbf{X}$ and a diagonal matrix $\mathbf{V}$ with elements $\widehat{\pi}_i(1-\widehat{\pi}_i)$ the Wald test is defined as (Hosmer and Lemeshow, 2000)

$$W = \widehat{\boldsymbol{\beta}}'(\mathbf{X}'\mathbf{V}\mathbf{X})\widehat{\boldsymbol{\beta}} \tag{4.68}$$

This tests for the $p+1$ coefficients equalling zero. The Wald test must be viewed with caution as Hauck and Donner (1977) showed that it behaved unusually and concluded that the likelihood ratio test should be used.

Both the likelihood ratio test and the Wald test have a chi-square distribution (Hosmer and Lemeshow, 2000) with $p+1$ degrees of freedom.

Interpretation of the Regression Coefficients

The odds ratio (Ψ) is used to interpret the regression coefficients. For continuous independent variables the log of the odds ratio for a particular variable is the change of c units of x giving a logit difference of (Hosmer and Lemeshow, 2000)

$$g(x+c) - g(x) = c\widehat{\beta}_i. \tag{4.69}$$

This gives an odds ratio of

$$\Psi(c) = e^{c\widehat{\beta}_i} \tag{4.70}$$

with associated confidence interval

$$e^{\left(c\widehat{\beta}_i \pm z_{\alpha/2} c\widehat{SE}(\widehat{\beta}_i)\right)}. \tag{4.71}$$

The odds ratio measures how much more likely it is for an outcome to be present when the independent variable is changed from x to $x+c$.

Model Selection

Stepwise logistic regression was performed within SAS (SAS(R) Proprietary Software Release (8.1)) where variables were entered and removed by their significance in the model and the process stopped when no more variables could be entered or removed.

	Group	Components
Female	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	None
	G4 G3 G2 vs G1	F1SCL
Male	G4 vs G3 G2 G1	None
	G4 G3 vs G2 G1	F1SCL F2SCL
	G4 G3 G2 vs G1	F1SCL F2SCL

Table 4.31: Stepwise logistic regression results using the likelihood ratio test.

Parameter	Estimate (SE)	Odds Ratio (90% CI)
Intercept	1.89 (0.70)	
F1 SCL	-0.24 (0.14)	0.79 (0.62, 1.00)

Table 4.32: Parameter estimates for the logistic regression model for the depressed females change in Hamilton score using the lower quartile as the cutpoint.

4.5.4 Results from the Logistic Regression

From the discriminant analysis the models of interest are those that are predicting the change in Hamilton score so these models were investigated with logistic regression. Rather than biasing the stepwise process by only using the models found in the discriminant analysis all the components were entered at the beginning to see if the same models were achieved as the discriminant analysis. The same models were found with the logistic regression. These models are shown in Table 4.31.

Logistic Regression for the Depressed Females Change in Hamilton Score using the Lower Quartile as the Cutpoint

The logistic regression model used F1SCL as the independent variable and was significant using the likelihood ratio test ($\chi^2 = 2.76$, $df = 1$, p-value= 0.0967) but not significant according to the Wald test ($\chi^2 = 2.67$, $df = 1$, p-value= 0.10) using a 10% level of significance. The odds ratio is 0.790 with a 90% confidence interval of (0.62, 1.00). The model parameters are shown in Table 4.32

The odds ratio confidence interval includes one so is not significant but the point estimate is less than one indicating that as the value of F1 SCL increases the odds of being a poor improver decreases. For example a F1 SCL score of zero corresponds to high odds of not improving at 6.618, and in contrast if F1SCL = 4 then the odds of not improving decreases to 2.58. This situation is illustrated in Figure 4.27. For every one

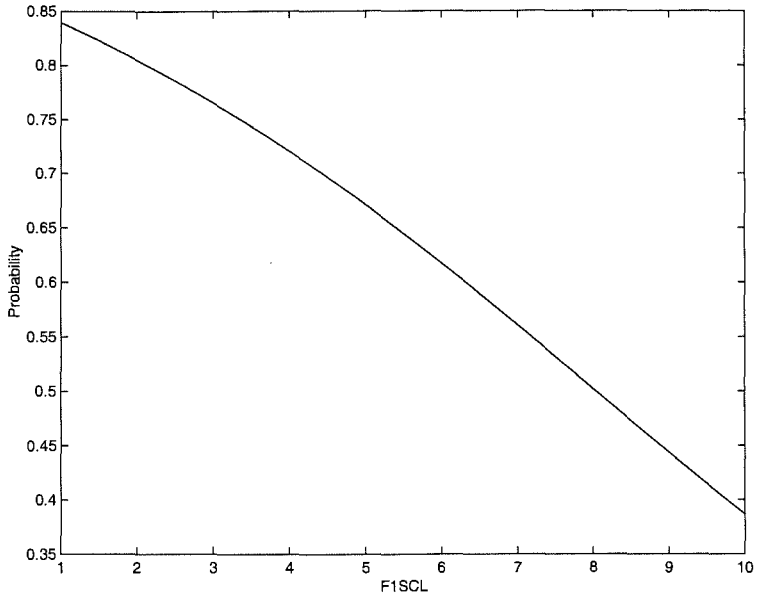


Figure 4.27: Predicted probabilities for F1SCL for the depressed females change in Hamilton score using the lower quartile as the cutpoint.

Parameter	Estimate (SE)	Odds Ratio (90% CI)
Intercept	2.32 (1.13)	
F1 SCL	-0.95 (0.42)	0.39 (0.19, 0.77)
F2 SCL	0.74 (0.40)	2.10 (1.09, 4.08)

Table 4.33: Parameter estimates for the logistic regression model for the depressed males change in Hamilton score using the lower quartile as the cutpoint.

unit increase in F1SCL score, the associated risk of being a poor improver decreased by 0.790.

Logistic Regression for the Depressed Males Change in Hamilton Score using the Lower Quartile as the Cutpoint

The logistic model for the depressed males using the lower quartile as the cut point found that F1SCL and F2SCL were significant predictors of the change in Hamilton score (likelihood ratio test $\chi^2 = 6.30$, $df = 2$, p-value$= 0.0429$). The model parameters are presented in Table 4.33.

F1 SCL has an odds ratio of 0.387 (0.194, 0.772) and F2 SCL has an odds ratio of 2.104 (1.085, 4.080). Neither of these intervals include one so both are significant. The odds ratio for F1 SCL is less than one so an increase in F1 SCL gives a decrease in the

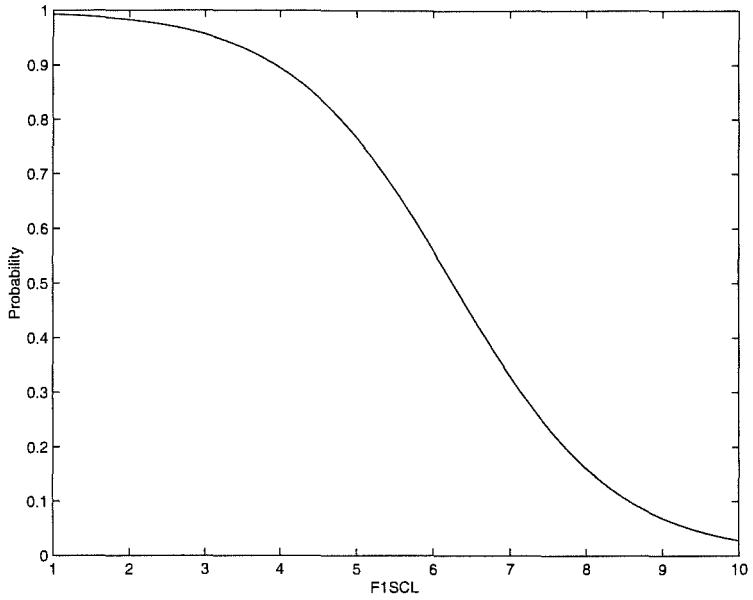


Figure 4.28: Predicted probabilities for F1SCL for the depressed males change in Hamilton score using the lower quartile as the cutpoint.

odds of being a poor improver. F2 SCL has a confidence interval larger than one so as F2 SCL increases there is an increase in the odds of being a poor improver. For example, holding F2 SCL fixed at its mean value (4.87), an F1 SCL score of one corresponds to high odds of not improving at 147, and in contrast if F1SCL is equal to 10 then the odds of not improving decreases to 0.29. This situation is illustrated in Figure 4.28. For every one unit increase in F1SCL score, the associated risk of being a poor improver decreased by 0.39. Holding F1 SCL fixed at its mean (4.97), an F2 SCL score of one corresponds to low odds of not improving at 0.1903, and if F2 SCL is equal to ten, then the odds of not improving increases to 154. This situation is illustrated in Figure 4.29. For every one unit increase in F2 SCL the risk of being a poor improver increases by 2.10.

Logistic Regression for the Depressed Males Change in Hamilton Score using the Median as the Cutpoint

The model for the median cut point is very similar to that of the upper quartile. The model involved F1 SCL and F2 SCL as the independent variables. The model was significant with a likelihood ratio test chi-square of 15.30 with 2 degrees of freedom leading to a p-value of 0.0005. The model parameters are presented in Table 4.34.

As with the lower quartile model the odds ratio for F1 SCL is below one (0.221 (0.097, 0.505)) and F2 SCL has an odds ratio above one (2.810 (1.399, 5.645)). This leads us to the same conclusion that an increase in F1 SCL leads to a decrease in the odds of being

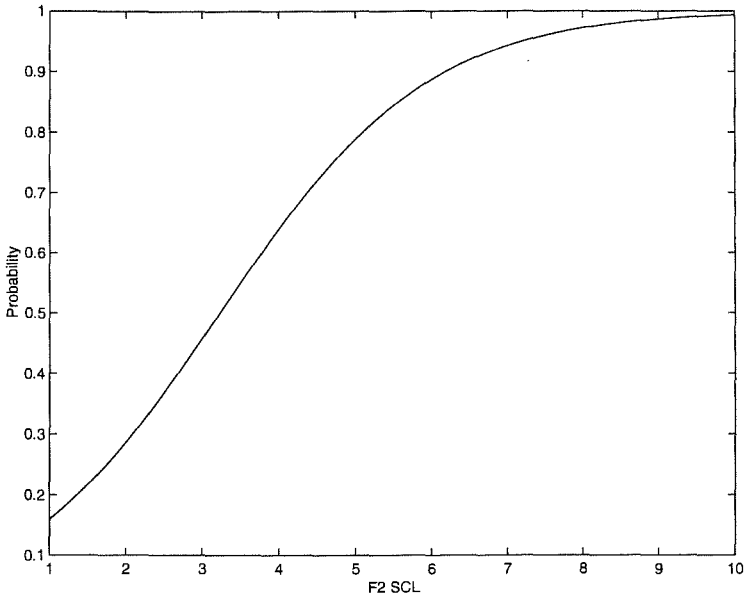


Figure 4.29: Predicted probabilities for F2SCL for the depressed males change in Hamilton score using the lower quartile as the cutpoint.

Parameter	Estimate (SE)	Odds Ratio (90% CI)
Intercept	2.32 (1.18)	
F1 SCL	-1.51 (0.50)	0.22 (0.10, 0.51)
F2 SCL	1.03 (0.42)	2.81 (1.40, 5.65)

Table 4.34: Parameter estimates for the logistic regression model for the depressed males change in Hamilton score using the lower quartile as the cutpoint.

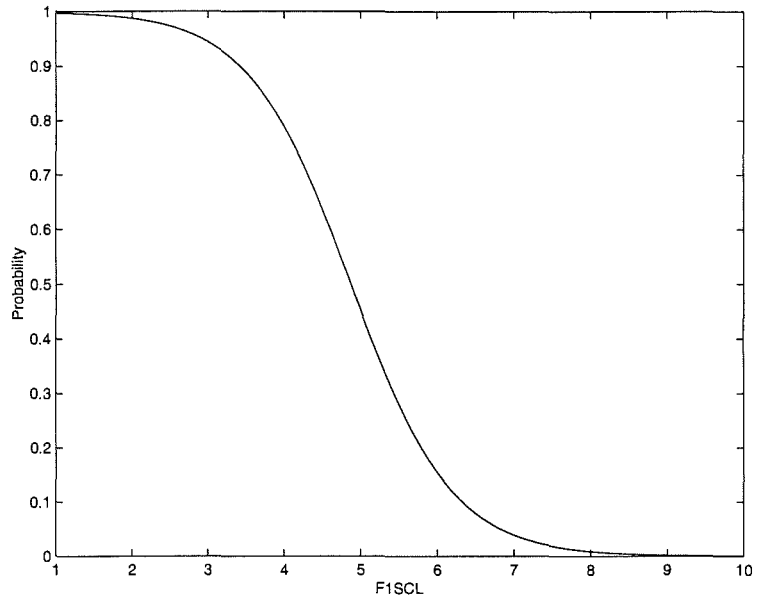


Figure 4.30: Predicted probabilities for F1SCL for the depressed males change in Hamilton score using the median as the cutpoint.

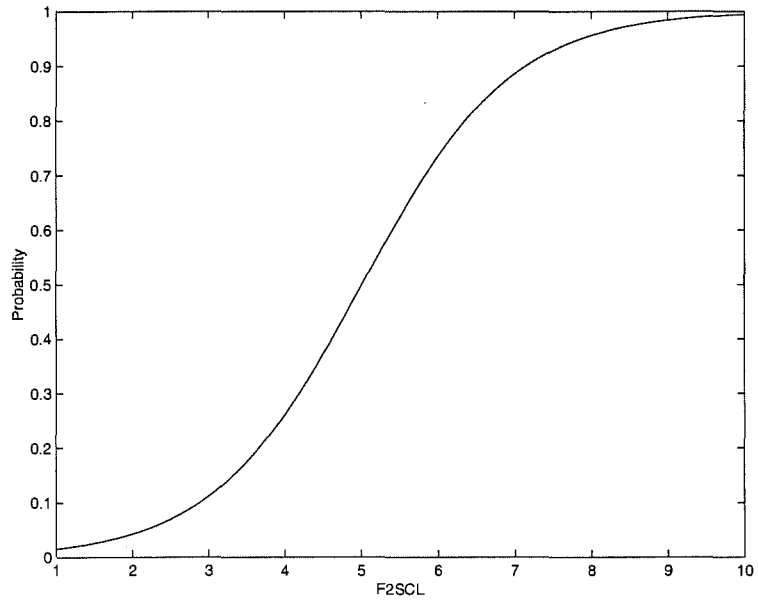


Figure 4.31: Predicted probabilities for F2SCL for the depressed males change in Hamilton score using the median as the cutpoint.

a poor improver. As F2 SCL increases the odds of being a poor improver also increase. For example, holding F2 SCL fixed at its mean value (4.87), an F1 SCL score of one corresponds to high odds of not improving at 346, and in contrast if F1SCL is equal to 10 then the odds of not improving decreases to 0.0004. This situation is illustrated in Figure 4.30. For every one unit increase in F1SCL score, the associated risk of being a poor improver decreased by 0.22. Holding F1 SCL fixed at its mean (4.97), an F2 SCL score of one corresponds to low odds of not improving at 0.0158, and if F2 SCL is equal to ten, then the odds of not improving increases to 173. This situation is illustrated in Figure 4.31. For every one unit increase in F2 SCL the risk of being a poor improver increases by 2.81.

4.6 Summary

This chapter has found the best of the structural models developed in Chapter 3, using confirmatory factor analysis on a second dataset. The models were then bootstrapped to counter normality issues and provide confidence intervals on the parameters and fit indices.

This is the first study to investigate the structural model underlying the seven Cloninger traits. This chapter has shown that consistently one factor is needed to best model the personality data. Thus the seven Cloninger traits have been reduced to a single construct representing harm avoidance against persistence, self directedness and either cooperativeness or self transcendence.

There is debate as to how many components the SCL questionnaire actually measures. Some studies, such as those by Carpenter and Hittner (1995), Bonynge (1993) and Bernstein et al. (1994), have shown evidence of a single overall factor of general distress, rather than nine distinct symptoms as presented by Derogatis and Cleary (1977). Bernstein et al. (1994) suggested a second factor might be appropriate, separate from the overall distress measure. Steer et al. (1994) found an overall general component of distress and identified four specific residual components that were appropriate for their study. Studies such as those by Vassend and Skrondal (1999) and Schwarzwald et al. (1991) presented evidence for more than one factor.

A second point of contention with the symptom checklist is the presence or absence of gender differences in the symptom structure. Bonynge (1993) showed gender invariance in a group of suicidal adults and adolescents. Vassend and Skrondal (1999); Carpenter and Hittner (1995) both showed significant gender differences, on data from, in the first case, the general population and data from psychiatric patients.

This study has found that when the depressed patients are severely depressed six factors are needed to describe the symptoms adequately, however after treatment when

symptoms have reduced substantially only two factors (for the females) are needed. This in part explains the differences seen in the literature for the number factors the SCL questionnaire measures. Studies with severely ill patients may find more factors than those with normal or less severely ill patients. Unfortunately due to sample size restrictions a definite answer on gender differences in the SCL questionnaire was unable to be answered. The Flury test from Chapter 3 suggested that the severely depressed patients (i.e. before treatment) had no gender differences in the underlying structure. However after treatment there were significant gender differences. Multigroup structural equation modelling found significant differences across gender at both time points. Further analysis on a larger sampler would be needed to answer this question more fully.

The longitudinal analysis found that the female baseline and post treatment personality structures had a stability across the factor scores. The only difference between the models was the replacement of cooperativeness after treatment by self transcendence.

The males personality however, was not stable across time. The factor scores decreased after treatment indicating that either harm avoidance decreased or persistence, self directedness or cooperativeness increased, or both occurred. The positive covariance, for both the male and female models, across time showed that those high on the factor tended to remain high on the factor and vice versa.

The female longitudinal model showed a decrease in symptoms. The positive covariance across time showed that those high in symptoms at baseline remained comparatively high in symptoms after treatment.

Table 4.35 presents the satisfactory models found with method used to calculate the model in the second column and the method that gave that number to rotate in the third column. There are eight models in total and ICA was the best method in five of those models. This includes the one model where IC had the same solution as PCA and FA. PCA was successful in three models and factor analysis in two. ICA was by far the best method to use. This is not surprising as ICA takes component analysis to the next level by striving for independence of components rather than decorrelated components, as is the case in PCA. Factor analysis was expected to do better than PCA as factor analysis is based on finding the type of model that is used in confirmatory factor analysis. PCA actually did slightly better.

The best method for number of components to rotate was AutoScree, which was correct in five models, followed closely by Velicer's MAP in four models. As either Velicer's MAP or Autoscree were right in all models these two methods were sufficient for this study to calculate the number of components. Any future studies will use these two measures to calculate the number of components to retain, however a further test will still be needed such as CFA to choose between the two methods if they disaggree.

Discriminant analysis and logistic regression were used to investigate the interpretabil-

Variable	Model	Number to Rotate Method
F TCI B	1 FA	Armor theta, Velicer MAP
F TCI E	1 IC	Armor theta, Velicer MAP, Auto Scree
M TCI E	1 PC/IC/FA	Armor theta, Velicer MAP
M & F SCL B	6 PC	% Var, Auto Scree
F SCL E	2 IC	Velicer MAP
F TCISCL B	7 IC	% Var, Auto Scree
	10 IC	% Var, AutoScree
M TCISCL B	6 PC	Auto Scree

Table 4.35: Comparison of component methods and number to rotate methods

ity of the components developed in this chapter. Both methods lead to the same independent variables. For the females, when the cut point in the change in Hamilton score was chose to be the lower quartile, F1 SCL was shown to have a protective effect. As F1SCL increases there is lease chance of being in the group of poor improvers (those people whose Hamilton score has improved the least from baseline to after treatment).

The males showed significant models for two of the cut points. The cut point for the lower quartile and the median gave similar models. For these cut points in the change in Hamilton score, F1 SCL and F2 SCL were the independent variables. F1 SCL was found to have a protective effect similar to that shown for the females. F2 SCL however was found to have a risk effect. The higher the levels of F2SCL the more likely to be a poor improver.

The first symptom component is a weighted average of somatisation, anxiety and psychotocism. The second symptom component is a weighted average of interpersonal sensitivity and paranoid ideation. Thus the results above suggest that for the females high levels of somatisation, anxiety and psychotocism lead to a decreased risk of being a poor improver. For the males high levels of somatisation, anxiety and psychotocism lead to a decreased risk of being a poor improver and high levels of interpersonal sensitivity and paranoid ideation lead to an increased risk of being a poor improver.

Future work will extend the confirmatory factor analysis to allow more complicated models. In particular for the males at times no model was found that described the covariance structure sufficiently, however the models were restricted to allowing variables to load on one factor only. This may not be the case in reality and more complex models allowing cross loading of variables may be better at describing the covariance structure.

The content of this chapter either looked at symptoms or personality separately to find the underlying structure or combined the two and allowed the factors to covary. Then these factors were investigated as predictors and discriminators of depression outcome.

However it is of interest to see if the symptoms can predict the personality types. The next chapter extends these models to path analysis with latent variables where the underlying symptom factors are predictors of the underlying personality factors and vice versa.

Chapter 5

Investigation of the Relationship Between Personality and Symptoms of Depression using Path Analysis and General Additive Models

The link between personality and symptoms of depression has been of interest for a number of years. A recent study (Grucza et al., 2003) used canonical correlation analysis and logistic regression to show significant relationships between depression symptoms and the four TCI temperament traits. They found significant relationships, particularly from harm avoidance, reward dependence and novelty seeking to certain depression symptoms. This chapter extends the work of Grucza et al. (2003) by investigating all seven of the Cloninger personality traits in relation to depression symptoms and by allowing for non-linear relationships.

There are two possible directions for the relationship, either symptoms predicting personality or personality predicting symptoms. The original questions posed for this thesis were mixed in directionality. The literature tends to investigate personality variables as predictors of symptoms. Both directions are investigated in this chapter. First the symptom variables are used to predict personality, then the personality variables are used as predictors of symptoms and the two directions are compared. The results are compared to the original hypotheses, and to the literature.

So far this thesis has investigated the latent structure of personality and symptoms ignoring any relationship between the two. This chapter will investigate the link between personality and symptoms of depression using two methods.

The first method is an extension to the confirmatory factor analysis used in Chapter 4. The best personality and symptom model at each time point will be modelled together,

with regression terms linking the personality factors to the symptoms factors and vice versa. This type of modelling is known as path analysis with latent variables and is part of the structural equation modelling framework (Bollen, 1989). The confirmatory factor analysis on the depressed males was unsuccessful at finding a reasonable model to describe their baseline personality and post treatment symptoms. Thus the path analysis was not able to be performed on the depressed males. The results for the depressed females are presented in Sections C.1 and 5.4.1.

The second method for investigating the link between personality and symptoms of depression is general additive models (GAMs) (Hastie and Tibshirani, 1990). These models extend multiple regression to non-linear modelling without prespecifying the type of non-linearity. This method will allow us to detect not only linear relationships between symptoms and personality, and vice versa, but also any non-linear relationships. The method was conducted on the personality traits and depression symptoms and for the females, the factors from the CFA models were also investigated to allow comparison with the path analysis models. Model building developed the best GAM models for each personality trait. There were a number of significant linear and non-linear relationships between personality and symptoms. Analysis of the residuals, via the R^2 and R^2_{adj} measures, revealed that whilst significant relationships existed generally the symptoms were poor predictors of personality and vice versa. However, 14 models had an R^2_{adj} greater than 30%.

The two methods are quite different and cover two important areas. The first method of path analysis allows for the modelling of relationships between the latent underlying factors rather than the manifest variables (the traits and symptoms). However, the draw back of this method is that the relationship is modelled linearly. The GAMs method introduces non-linear modelling but does not allow for modelling of the latent structure. The factors can be investigated but are not modelled as latent variables that relate to the traits and symptoms. The literature to date has tended to use standard regression and repeated measures analysis of varaince (Joffe et al., 1993; Joyce et al., 1994a).

The following hypotheses, that were proposed by Peter Joyce (personal correspondence), will be discussed and compared to the results presented in this chapter.

1. Depressed patients who have a high total score (or high on the first big global factor) will have high harm avoidance score and low self directedness.

2. Depressed patients with a high anxiety score will also have high harm avoidance.

3. Depressed patients with a high anger hostility score will have high novelty seeking and low cooperativeness.

4. Depressed patients with a high obsessive compulsive score will have low novelty seeking, high harm avoidance, low reward dependence and high persistence.

5. Depressed patients with a high somatisation score will be high on novelty seeking, harm avoidance, reward dependence and persistence. This could be gender specific.

Joyce and Paykel (1989) presented a review of the prediction of depression outcomes. At that time the variables investigated showed poor relationships with depression outcomes and Joyce and Paykel (1989) suggested that biological indicators would need to be studied. During the 1980s and 1990s personality has been investigated in relation to depression and the biologically based Cloninger model has shown more promise (Nelson and Cloninger, 1995). Joffe and Regan (1988) and Hirschfeld et al. (1983b) showed that personality scores can be altered by the depressed stated. Svrakic et al. (1992) showed harm avoidance could be related to the mood symptoms in normal volunteers.

Investigations into personality questionnaires other than the Cloninger model have demonstrated some of the following results. The personality characteristics of neuroticism and extraversion have been studied in relation to depression by groups such as Frank et al. (1987), Hirschfeld and Klerman (1979), Hirschfeld et al. (1983a), Hirschfeld et al. (1983b), Hirschfeld et al. (1989), Kerr et al. (1970), Liebowitz et al. (1979) and Weissman et al. (1978). In general the studies show that higher neuroticism scores are found in depressed patients than controls. Neuroticism scores decrease slowly with improvement of the depression symptoms. The results for extraversion vary more from study to study, however low extraversion generally related to more severe depression. These results in further detail are presented in Enns and Cox (1997).

Personality models have been developed that measure the personality characteristics most associated with depression (Akiskal and Hirschfeld, 1983; Beck et al., 1983). The relationships between these characteristics and depression have been studied, for example by Hirschfeld et al. (1976), Hirschfeld et al. (1977), Hirschfeld et al. (1986), Rohde et al. (1990), Boyce and Parker (1989), Blatt (1974) and Frank et al. (1987). Enns and Cox (1997) summarise the findings from these studies in their review paper.

The most recent study, as far as the author is aware, to investigate the Cloninger temperament model in relation to depression is presented in Grucza et al. (2003). Grucza et al. (2003) investigated the relationship between the temperament dimensions (novelty seeking, harm avoidance, reward dependence and persistence) and depressive symptoms measured by the Center for Epidemiologic Studies Depression Scale (Radloff, 1977) in a sample of 804 adults from the general population. The study used canonical correlation analysis and logistic regression. The first personality canonical correlation component measured mainly harm avoidance and was positively correlated with a symptom component measuring an overall severity of symptoms. The second personality component

constituting reward dependence and persistence was positively correlated with a symptom component that mainly measured vegetative symptoms and dysphoria. Logistic regression was used to investigate other measures of depression in relation to the temperament measures.

The study by Grucza et al. (2003) concentrated on the temperament personality variables. Likewise most of the studies using the Cloninger personality model have concentrated on the temperament variables (Enns and Cox, 1997). Joffe et al. (1993) investigated novelty seeking, harm avoidance and reward dependence before and after treatment in unipolar depressed patients (n=40). They classified the patients into recovered and non-recovered and found, using repeated measures analysis of variance, that there was no significant effect between novelty seeking and depression outcome, and reward dependence and depression outcome. Harm avoidance had significantly higher values in non-responders than responders. This study had a small sample size and did not investigate a gender effect.

Mulder and Joyce (1994) examined novelty seeking, harm avoidance and reward dependence scores in relation to the Hamilton Depression Rating Scale. Of the three personality variables, harm avoidance was significantly correlated with the level of depression. They also examined the relationship between the personality traits and other personality models.

Perhaps the most convincing study was that conducted by Joyce et al. (1994b). They investigated the depression outcomes in relation to personality in 84 depressed patients. Rather than model just the temperament traits directly to the percentage improvement in depression, they dichotomised the three variables, novelty seeking, harm avoidance and reward dependence, into high and low to give eight temperament types. Using multiple regression techniques they found that these eight types accounted for 25% of the variance in treatment outcomes. Alongside these results they showed that for their sample, patients with high harm avoidance and reward dependence tended to have good outcomes regardless of gender or drug choice. The combination of low novelty seeking, harm avoidance and reward dependence also lead to good treatment outcomes.

Joyce et al. (1994a) used a sample of 94 depressed patients and 40 normal controls to investigate daily fluctuations in cortisol levels. Hypersecretion of cortisol is often found in depressed patients. They found that reward dependence was significantly correlated to cortisol level and novelty seeking was significantly related to morning cortisol levels.

Nelson and Cloninger (1995) used the four temperament variables, novelty seeking, harm avoidance, reward dependence and persistence to predict treatment outcomes in a small sample of depressed patients. They found that harm avoidance significantly correlated with the Hamilton Depression Rating and reward dependence significantly correlated with the percent change in the Hamilton score. Using multiple regression

techniques they found that reward dependence and harm avoidance, and their interaction, accounted for 37% of the variance of the percentage change.

The most recent studies investigate all seven of the Cloninger traits in relation to depression. Farmer et al. (2003) investigated the genetic vulnerability to develop depression and found that novelty seeking, harm avoidance, reward dependence and self directedness were related to depression. On another study (Agosti and McGrath, 2002) depressed patients were treated with fluoxetine, imipramine or placebo. Personality was measured before treatment and then 8 weeks later. The study found that those who responded to treatment had significantly reduced harm avoidance but still higher than the normal controls, and self directedness levels returned to normal. The two different drugs did not result in differences in personality traits except self transcendence.

Luty et al. (2002) investigated the relationship between interpersonal sensitivity and temperament and character. The results showed strong correlations between interpersonal sensitivity and both the temperament and character scores. Marijnissen et al. (2002) found that harm avoidance scores were significantly higher in depressed patients before and after treatment compared to normals. In their study the TCI scores were not predictive of response to treatment. In a study of 108 depressed patients (Hirano et al., 2002) the Hamilton depression rating was positively correlated to harm avoidance and negatively correlated to self directedness and cooperativeness. In the responders group these scores improved with symptom improvement. Dysfunctional attitudes in depressed patients were related to self directedness in a study by Luty et al. (1999).

Corruble et al. (2002) investigated the personality changes in patients recovering from depression. They found that early changes involved decreased harm avoidance, and increased self directedness and cooperativeness. Delayed changes included increased self directedness and decreased self transcendence. Naito et al. (2000) showed that self directedness could be predictive of depression. Sato et al. (1999) used logistic regression to predict drug response from TCI personality scores. They found that the character traits self directedness and cooperativeness were important. Hansenne et al. (1999) found that self directedness and cooperativeness were related to depression severity using the Hamilton scale.

5.1 Path Analysis with Latent Variables

The theory for path analysis was introduced in section 4.1. The full model will be used so that the relationship between symptoms and personality, and vice versa, will be described by

$$\eta = \Gamma\xi + \zeta, \tag{5.1}$$

where η are the latent personality variables, ξ are the latent symptom variables with regression coefficients Γ and error ζ. The predicted variables (y) depend on their latent factors via

$$y = \Lambda_y \eta + \epsilon, \tag{5.2}$$

and the predictor variables (x) depend on their underlying factors via

$$x = \Lambda_x \xi + \delta. \tag{5.3}$$

The relationship between the latent factors is linear, however there may be a non-linear relationship between personality and symptoms. The following section introduces the theory for general additive models, which model non-linear terms using smoothing functions. The use of the non-linear modelling technique will allow the relationship between personality and symptoms to be probed thoroughly. The only draw back is that full latent structure modelling cannot be performed.

5.2 General Additive Models

General additive models (GAMs) are a recent development that extends standard linear regression to automatically fit non-linear terms, the type of which, does not have to be prespecified. GAMs have been used in a number of fields such as ecological modelling (Guisan et al., 2002), environmental research (Axtell et al., 2000), political science (Beck and Jackman, 1998) and biostatistics (Dalrymple, 2003). Croudace et al. (2000) used GAMs to investigate the relationship between an index of social deprivation, psychiatric admission prevalence and the incidence of psychosis. As far as the author is aware GAMs have not been used to relate personality to symptoms and vice versa. The following section introduces the theory for GAMs.

GAMs are an extension to standard regression techniques. Multiple linear regression is of the form

$$Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \cdots + \beta_p X_p, \tag{5.4}$$

for p variables. This can be extended to specific non-linear cases if the non-linear function is known, for example

$$Y = \beta_0 + \beta_1 X_1 + \beta_2 X_1^2, \tag{5.5}$$

models a quadratic relationship between X_1 and Y. Generally the non-linear component will be unknown making standard regression techniques uninviting. General additive

models extend the regression to a non-linear form by allowing each regression variable to have a non-linear relationship with the dependent variable. This non-linearity is calculated and tested in the process. The general additive model is

$$Y = \beta_0 + f_1(X_1) + f_2(X_2) + \cdots + f_p(X_p), \tag{5.6}$$

or

$$Y = \beta_0 + \sum_{i=1}^{p} f_i(X_i). \tag{5.7}$$

where f are the non-linear smoothing functions estimated in a nonparametric manner. The smoothing functions are found using a back fitting algorithm, details of which can be found in Hastie and Tibshirani (1990). The back fitting, in essence, fits the smooth functions by looking at the residuals (Venables and Ripley, 1997)

$$Y - \sum_{p \neq j} f_p(X_p), \tag{5.8}$$

and smoothing against the other X_j using cubic smoothing splines. The smoothing spline is calculated as (Hastie and Tibshirani, 1990)

$$\sum [y_i - f(x_i)]^2 + \lambda \int (f''(x))^2 dx \tag{5.9}$$

for n (x_i, y_i) pairs. The smoothing spline works by fitting a series of piecewise polynomials that have break points (or knots) at the x_i's. The smoothing spline balances perfect fit against smoothness. Computation of the cubic spline starts by defining B_j spline basis functions with coefficients (γ_j) to give the smoothing function $S(x) = \sum_1^{n+2} \gamma_j B_j(x)$. Using this information and letting $\boldsymbol{\Omega_{ij}} = \int B_i''(x) B_j''(x) dx$ equation 5.9 is solved for f. Rewriting equation 5.9 gives

$$(\boldsymbol{y} - \boldsymbol{B\gamma})^{\boldsymbol{T}} (\boldsymbol{y} - \boldsymbol{B\gamma}) + \boldsymbol{\lambda \gamma^T \Omega \gamma}, \tag{5.10}$$

and

$$\frac{d}{d\gamma}((\boldsymbol{y} - \boldsymbol{B\gamma})^{\boldsymbol{T}} (\boldsymbol{y} - \boldsymbol{B\gamma}) + \boldsymbol{\lambda \gamma^T \Omega \gamma}) = (\boldsymbol{B^T B} + \boldsymbol{\lambda \Omega})\boldsymbol{\gamma} - \boldsymbol{B^T y}. \tag{5.11}$$

Setting equation 5.11 equal to zero and using the Cholesky factorisation $(\boldsymbol{B^T B} + \boldsymbol{\lambda \Omega}) = \boldsymbol{L L^T}$ gives

$$\boldsymbol{L L^T \widehat{\gamma}} = \boldsymbol{B^T y} \tag{5.12}$$

which can be solved for $\widehat{\gamma}$ and thus the spline function is computed. To fit all smoothing functions the back fitting algorithm (Hastie and Tibshirani, 1990) is used. This is an iterative procedure that finds the initial estimates, then uses the residuals, as in equation

5.8, to estimate the smoothing functions. The second step is repeated until there are no changes in the smoothing functions.

Not all terms in equation 5.6 necessarily have to have a non-linear relationship. To model both a linear and non-linear relationship the semi-parametric model is used, as follows

$$y = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + f_3(x_3) + f_4(x_4). \tag{5.13}$$

5.2.1 Measures of Parameter and Model Fit

For the parametric (linear) parts of the model, the significance of the regression coefficients can be tested using (McClave and Sincich, 2000)

$$t = \frac{\widehat{\beta}_2}{s_{\widehat{\beta}_2}}. \tag{5.14}$$

A significant result suggests that the slope for that variable is significantly different from zero (or some hypothesised value). For parametric models this can be extended to a test of all coefficients being significantly different to zero by use of the following F-test

$$F = \frac{(SS_{yy} - SSE)/k}{SSE/(n-(k+1))}, \tag{5.15}$$

where SSE is the variance not measured by the model

$$SSE = \sum (y - \widehat{y})^2 \tag{5.16}$$

and the variance of the dependent variable is

$$SS_{yy} = \sum y^2 - \frac{(\sum y)^2}{n}. \tag{5.17}$$

For a given model, $\widehat{\boldsymbol{\mu}}$, the deviance is defined as (Hastie and Tibshirani, 1990)

$$D(\mathbf{y}; \widehat{\boldsymbol{\mu}}) = 2l(\boldsymbol{\mu}_{max}; \mathbf{y}) - l(\widehat{\boldsymbol{\mu}}; \mathbf{y}) \tag{5.18}$$

where $l(;)$ is the log likelihood. This measure can be used for assessing how well the model fits because it is similar to the residual sum of squares in ordinary regression. The deviance for the full model is compared to the model without the spline of interest, to get an approximate F-test for the spline (Hastie and Tibshirani, 1990). This gives a significance test for the spline function analogous to the t-test above.

The models with non-linear terms will be further examined by use of the partial residuals. The partial residuals are defined in the back fitting function (Hastie and Tibshirani, 1990). The partial residuals for the variable of interest can be plotted after removal of

the effects of the other variables. The model terms are additive so effects from each of the other variables are easily removed. Residuals are examined for influential or outlying cases and non-random trends.

The GAMs procedure was conducted in SAS (SAS(R) Proprietary Software Release (8.2)) using a cubic spline smoothing function. The models assessed in Section C.2, used the symptom variables to predict the personality variables, as well as the factors, if available, for both symptoms and personality. Section 5.4.2 presents the models, using the personality variables to predict the symptom variables. Models were developed using the following approach. Ideally all combinations would be tested and compared for the lowest deviance, however, this would involve computing a large number of models as there are nine independent variables in the first instance. So first the significant x variables were found individually and used together as a base model. From this base model all the other x variables were retested for significance in the presence of the base model. This process was stopped when no further variables were found to be significant. If variables were found to be significant individually in the model but not when used together, the deviance was then used to find the best model.

Once the best models have been found a further diagnostic needs to be used to look at how well each model predicts the dependent variable. The R^2 statistic will be used to assess the goodness of fit of each model. The R^2 statistic measures the amount of variance of the dependent variable that is measured by the model. The variance not measured by the model can be measured using the sums of squares of the residuals

$$SSE = \sum (y - \widehat{y})^2 \tag{5.19}$$

and the variance of the dependent variable is given in equation 5.17. Thus the R^2 statistic is

$$R^2 = \frac{SS_{yy} - SSE}{SS_{yy}}. \tag{5.20}$$

The more complex the model, the better R^2 will be (McClave and Sincich, 2000), so this can be adjusted as follows,

$$R^2_{adj} = 1 - \left(\frac{(n-1)}{n-(k+1)}\right)(1 - R^2) \tag{5.21}$$

where n is the number of observations and k is the number of independent variables.

5.3 Predicting Personality from Symptoms of Depression

The results from the prediction of personality from symptoms of depression are presented in full in Appendix C. This directionality is of less interest clinically but statistically was still worth investigating. An overview of the results is presented below.

5.3.1 Overview

In general, symptoms are poor predictors of the personality traits even though there are significant relationships between them. The two structural models analysed, showed poor fit and insignificant loadings for the regression terms between the latent symptom and personality variables. The path analysis model is superior to multiple linear regression as it allows for the modelling of the latent structure. However, the draw back of path analysis is that it only models linear terms. The results from the path analysis show that there are no significant relationships from the symptom model to the personality model at both time points for the depressed females. The depressed males were not analysed in this manner, as at baseline, only a symptom model was available and after treatment only a personality model. The path analysis agrees with the results found in Appendix B where combined TCI and SCL models were developed. These models had few personality variables in them and when they were involved in the model, the factors did not have significant covariances with the symptom factors.

To thoroughly investigate any potential relationship from symptoms to personality, general additive models (GAMS) were used. There were a number of significant linear and non-linear relationships between personality and symptoms. Analysis of the residuals, via the R^2 and R^2_{adj} measures, revealed that whilst significant relationships existed the symptoms were poor predictors of personality.

The best model found was for the depressed females after treatment (Model 3, Table C.4). Approximately 45% of the variance in harm avoidance could be explained by interpersonal sensitivity, depression, anger hostility, paranoid ideation and obsessive compulsive symptoms. The first four symptoms were linear predictors, whilst obsessive compulsion was non-linear. Low levels of interpersonal sensitivity, depression and high levels of anger hostility, paranoid ideation and obsessive compulsive predict low levels of harm avoidance.

Two other models (Model 6, Table C.3 and Model 6, Table C.4) were able to explain approximately 30% of the variance of a particular trait, whilst the other models were all below 30%. This suggests that whilst some general trends can be established, i.e. for depressed females post treatment high interpersonal sensitivity scores relate to low self

directedness scores; actual prediction of personality traits from symptoms will be poor and manifest large errors.

The significant relationships found are summarised as follows. The baseline females have the following significant relationships with the symptom variables, models with an $R^2_{adj} > 0.30$ are shown in bold.

$$F1_{TCI} = -0.22F1_{SCL} + 0.47F2_{SCL} + 0.28F3_{SCL} + 0.21F4_{SCL} - 0.87$$

$$NS_{TCI} = 0.03AH_{SCL} + 0.48$$

$$HA_{TCI} = 0.14IS_{SCL} - 0.07PI_{SCL} + 0.55$$

$$RD_{TCI} = none$$

$$P_{TCI} = -0.07IS_{SCL} + 0.05S_{SCL} + 0.54$$

$$\mathbf{S_{TCI} = 0.04S_{SCL} - 0.09PI_{SCL} - 0.10IS_{SCL} + 0.74}$$

$$C_{TCI} = -0.11PI_{SCL} + 0.07PA_{SCL} + 0.87$$

$$ST_{TCI} = 0.03OC_{SCL} + 0.05S_{SCL} + 0.20$$

The following models represent the males at baseline.

$$NS_{TCI} = none$$

$$HA_{TCI} = 0.10IS_{SCL} + 0.51$$

$$RD_{TCI} = 0.10A_{SCL} - 0.07S_{SCL} + 0.49$$

$$P_{TCI} = 0.06OC_{SCL} + 0.39$$

$$S_{TCI} = -0.06OC_{SCL} - 0.06IS_{SCL} + 0.71$$

$$C_{TCI} = -0.06AH_{SCL} - 0.08P_{SCL} + 0.05A_{SCL} + 0.85$$

$$ST_{TCI} = 0.07PI_{SCL} + f(IS_{SCL}) + f(PA_{SCL}) + 0.28$$

Models were developed for the post treatment data. The best models for the females are shown below.

$$F1_{TCI} = f(F2_{SCL})$$

$$NS_{TCI} = -0.11A_{SCL} + 0.07PI_{SCL} + f(S_{SCL}) + 0.52$$

$$\mathbf{HA_{TCI} = 0.17IS_{SCL} + 0.17D_{SCL} - 0.10AH_{SCL} - 0.19PI_{SCL} + f(OC_{SCL}) + 0.51}$$

$$RD_{TCI} = -0.07PI_{SCL} + f(S_{SCL}) + 0.76$$

$$P_{TCI} = -0.19S_{SCL} + f(PI_{SCL}) + f(PA_{SCL}) + 0.55$$

$$\mathbf{S_{TCI} = -0.15IS_{SCL} + f(P_{SCL}) + 0.75}$$

$$C_{TCI} = -0.03AH_{SCL} - 0.04PI_{SCL} + 0.88$$

$$ST_{TCI} = 0.07PI_{SCL} + 0.30$$

The post treatment models for the males are below.

$$NS_{TCI} = -0.06OC_{SCL} + 0.55$$

$$HA_{TCI} = 0.17IS_{SCL} + 0.47$$

$$RD_{TCI} = -0.07IS_{SCL} + 0.65$$

$$P_{TCI} = none$$

$$S_{TCI} = -0.14D_{SCL} + 0.73$$

$$C_{TCI} = -0.14P_{SCL} + f(PA_{SCL}) + 0.84$$

$$ST_{TCI} = -0.18PA_{SCL} + f(A_{SCL}) + 0.28$$

There appears to be more significant relationships post treatment between the symptoms and personality than at baseline. Two of the post treatment models for the females had R^2_{adj} values above 30% and only one baseline model had an R^2_{adj} above 30%, though this reduced, to below 30%, when a potential outlier was removed from the model. The baseline female models were all linear, where as the post treatment models are more complicated with non-linear terms in six of the eight models.

Interpersonal sensitivity was positively and linearly related to harm avoidance in both males and females at both time points. The models for cooperativeness are interesting. At baseline high values of anger hostility are related to low values of cooperativeness for both the males and females. After treatment anger hostility does not feature in the models, but phobic anxiety does for both males and females. For the females, paranoid ideation is negatively related to cooperativeness at both time points, and for the males psychotocism is negatively related to cooperativeness at both time points.

Interpreting the Three Models with $R^2_{adj} > 0.3$

From the female baseline models high self directedness is predicted by low somatisation, high interpersonal sensitivity and high psychotocism (Figure C.5). For the females after treatment two models have symptom variables that are reasonable predictors of the two personality traits. The first model has low levels of interpersonal sensitivity and depression and high levels of anger hostility and paranoid ideation predicting high levels of harm avoidance (Figure C.6). This model also includes a non-linear term. The spline curve of Figure C.8 shows that the maximum values of harm avoidance occur for values of obsessive compulsive around 0.4, values above or below this have lower values of harm

avoidance. The second model predicts high self directedness levels for low interpersonal sensitivity scores (Figure C.9) and the spline curve (Figure C.11) suggests that self directedness values decrease as psychotocism increases up to a value of about 0.4 then the self directedness values increase with increasingly psychotic symptoms.

The Depression Symptom Predicting Personality

The study involves a dataset of depressed patients so the most relevant symptom to them is depression. Depression was a predictor of personality in two models. Both of these models were for the post treatment data. In the first model depression was one of five symptoms predicting the female's harm avoidance scores. The relationship between depression and harm avoidance was positive and linear. Low levels of depression predicted low levels of harm avoidance. In the second model depression is the single predictor of self directedness and the relationship is linear and negative. Thus, low depression levels predicted high self directedness levels.

5.4 Predicting Symptoms from Personality

The first section has dealt with the prediction of personality variables from the symptom variables. This section investigates personality variables as predictors of symptoms using the methods, as before, of path analysis and GAMs. This directionality matches that seen in the literature.

5.4.1 Path Analysis Results

Depressed Females at Baseline

Path analysis was again conducted on the female's baseline symptom and personality models, this time personality was used to predict the symptoms. The path diagram is presented in Figure 5.1. The model was bootstrapped in SAS (SAS(R) Proprietary Software Release (8.1)) to obtain confidence intervals on the parameters.

The regression coefficients from the latent personality factor to the latent symptom factors are not significantly different from zero because the confidence interval contains zero. Likewise the loadings of the personality variables onto the latent factors are all zero.

The fit indices presented in Table 5.1 suggest very poor model fit. Both under naïve bootstrapping and with the Bollen-Stine transformation, none of the indices are in the appropriate bounds. This model does not represent the relationship, if any, from personality to symptoms for the baseline females.

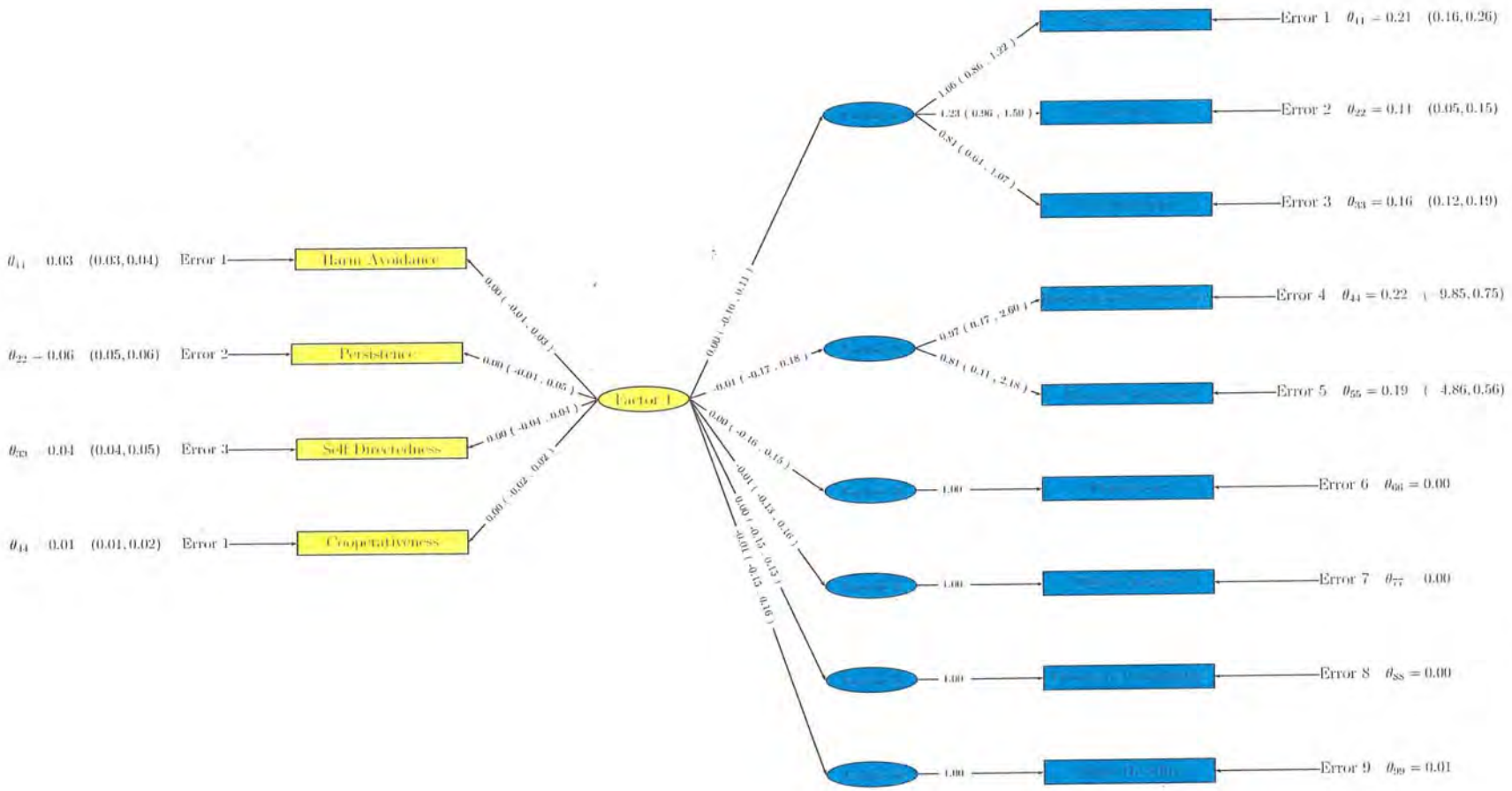


Figure 5.1: Depressed females personality predicting symptoms at baseline.

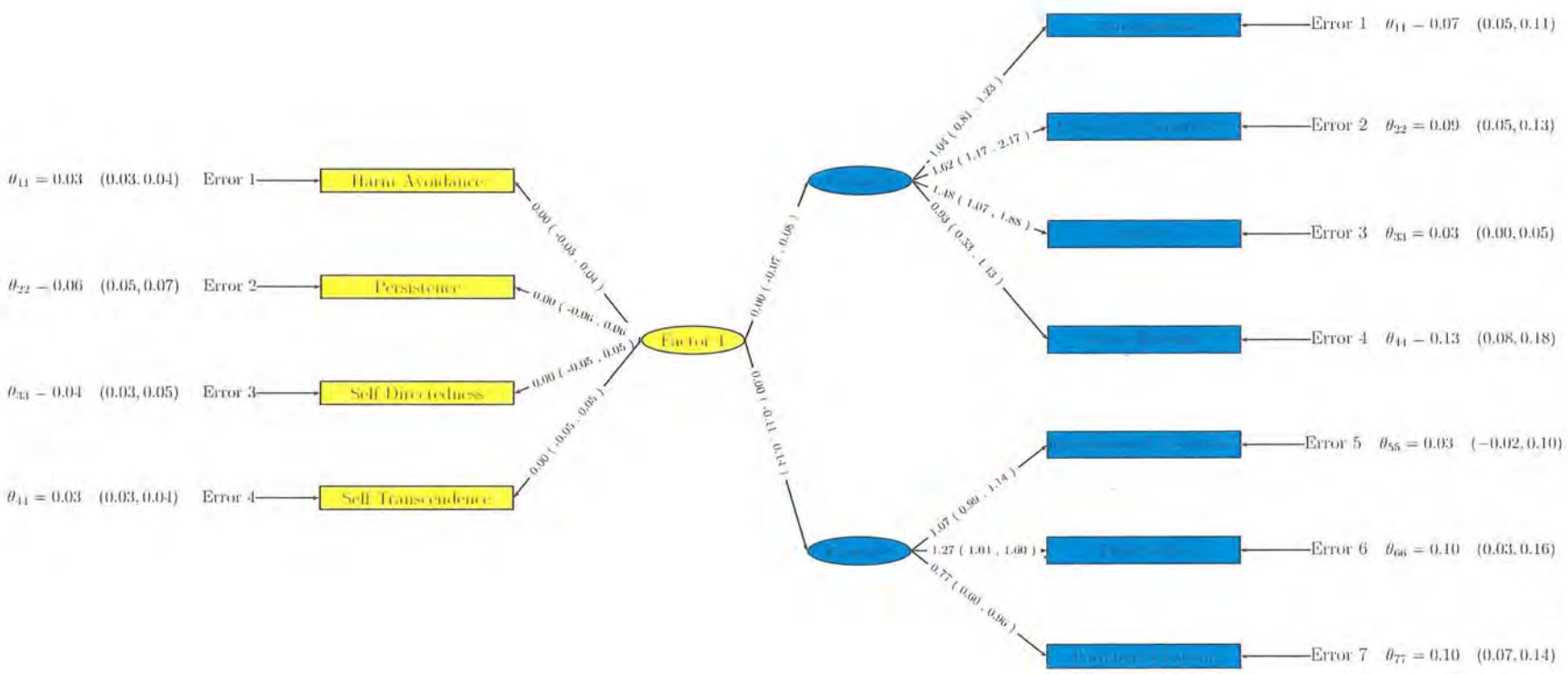


Figure 5.2: Depressed females personality predicting symptoms post treatment.

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.474	(0.437, 0.517)	0.445	(0.410, 0.485)
AGFI	0.254	(0.202, 0.315)	0.212	(0.163, 0.269)
RMR	0.164	(0.137, 0.193)	0.169	(0.143, 0.197)
Chi-Square	1192	(1059, 1327)	1096	(959, 1239)
df	74		74	
p-value	0.000	(0.000, 0.000)	0.000	(0.000, 0.000)
RMSEA	0.261	(0.245, 0.277)	0.250	(0.233, 0.267)
CFI	0.272	(0.233, 0.311)	0.142	(0.104, 0.187)
AIC	1043	(911, 1179)	948	(811, 1091)
NFI	0.267	(0.231, 0.305)	0.145	(0.110, 0.187)

Table 5.1: Fit indices for the baseline female's path analysis model.

Depressed Females after Treatment

A similar situation is found for the depressed females after treatment. There are no significant relationships between the latent personality factor and the latent symptom factors (Figure 5.2). The personality variables have loadings equal to zero, on the latent factor and the fit indices, presented in Table 5.2, are an improvement on the baseline model but still indicate poor fit with no indices in the appropriate bounds. Either there is no relationship from personality to symptoms or if there is, path analysis is the wrong tool to describe it, or the structural model is defined inadequately.

Overview of Path Analysis

These results match those presented in Section C.1 where the symptoms were used as predictors of personality. No relationship was found using that directionality. These results also support the results presented in Section B. The structural models that combined personality and symptoms showed a dominance of symptoms and generally any personality factors present did not covary with the symptom factors.

5.4.2 The Best General Additive Models

Using the same model building process as in Section 5.2 the best models for predicting the symptoms from the personality variables were developed and are presented in Tables 5.3 to 5.6. The following section discusses these models before an investigation of the residuals is conducted in Section 5.4.3.

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.661	(0.623, 0.697)	0.714	(0.675, 0.751)
AGFI	0.481	(0.424, 0.537)	0.563	(0.503, 0.619)
RMR	0.080	(0.059, 0.104)	0.076	(0.055, 0.101)
Chi-Square	498	(432, 573)	390	(329, 463)
df	51		51	
*p*value	0.000	(0.000, 0.000)	0.000	(0.000, 0.000)
RMSEA	0.256	(0.236, 0.276)	0.223	(0.202, 0.246)
CFI	0.547	(0.48 , 0.601)	0.594	(0.534, 0.648)
AIC	396	(330, 471)	288	(227, 361)
NFI	0.527	(0.469, 0.578)	0.567	(0.510, 0.618)

Table 5.2: Fit indices for the post treatment female's path analysis model.

The Females at Baseline

Table 5.3 presents the best models for the females at baseline. The models contain mainly the character traits with self directedness and self transcendence important in most models, in fact self transcendence is in every model. Of the temperament traits novelty seeking and harm avoidance appear in some of the models. Reward dependence and persistence do not appear at all and are not important in the prediction of the female's symptoms at baseline.

The Females After Treatment

The depressed females after treatment have four important variables in the final GAMs presented in Table 5.4. Similarly to the baseline results, self directedness and self transcendence are important, but after treatment harm avoidance and reward dependence are also important. All the personality variables feature in at least one model.

Comparison of the Females Before and After Treatment

The symptom factors are significantly related to the personality factor at both time points. The path analysis found no significant relationships between the symptom factors and the personality factor, however, the path analysis model measures all these relationships simultaneously and in a latent modelling environment.

Harm avoidance and self directedness are significantly related to somatisation at both time points. At baseline novelty seeking is also involved in the model; after treatment reward dependence and persistence are involved in the model. Obsessive compulsive

Model Number	Symptom Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
FB 1	Factor 1 SCL	Factor 1 TCI	
FB 2	Factor 2 SCL	Factor 1 TCI	
FB 3	Factor 3 SCL	Factor 1 TCI	
FB 4	Factor 4 SCL	Factor 1 TCI	
FB 5	Factor 5 SCL		Factor 1 TCI
FB 6	Factor 6 SCL	Factor 1 TCI	
FB 7	Somatisation	Harm Avoidance	Novelty Seeking
		Self Transcendence	
FB 8	Obsessive Compulsive	Self Transcendence	Self Directedness
FB 9	Interpersonal Sensitivity	Self Directedness	Harm Avoidance
		Self Transcendence	
FB 10	Depression	Self Transcendence	Self Directedness
FB 11	Anxiety	Harm Avoidance	
		Self Directedness	
		Self Transcendence	
FB 12	Anger Hostility	Self Directedness	
		Cooperativeness	
		Self Transcendence	
FB 13	Phobic Anxiety	Harm Avoidance	
		Cooperativeness	
		Self Transcendence	
FB 14	Paranoid Ideation	Cooperativeness	Harm Avoidance
		Self Transcendence	Self Directedness
FB 15	Psychotocism	Self Directedness	
		Cooperativeness	
		Self Transcendence	

Table 5.3: Best model from GAMS for the depressed females at baseline.

Model Number	Symptom Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
FP 1	Factor 1 SCL	Factor 1 TCI	
FP 2	Factor 2 SCL		Factor 1 TCI
FP 3	Somatisation	Harm Avoidance	
		Reward Dependence	
		Persistence	
		Self Transcendence	
FP 4	Obsessive Compulsive	Harm Avoidance	
		Reward Dependence	
		Self Directedness	
		Self Transcendence	
FP 5	Interpersonal Sensitivity	Harm Avoidance	
		Self Directedness	
FP 6	Depression	Reward Dependence	Harm Avoidance
		Self Directedness	
		Self Transcendence	
FP 7	Anxiety	Harm Avoidance	
		Reward Dependence	
		Self Directedness	
		Cooperativeness	
		Self Transcendence	
FP 8	Anger Hostility	Self Directedness	
FP 9	Phobic Anxiety	Harm Avoidance	Persistence
FP 10	Paranoid Ideation	Reward Dependence	Cooperativeness
		Self Directedness	
		Self Transcendence	
FP 11	Psychotocism	Self Directedness	Novelty Seeking
		Self Transcendence	Reward Dependence

Table 5.4: Best model from GAMS for the depressed females post treatment.

symptoms are significantly related to self directedness and self transcendence at both time points. After treatment harm avoidance and reward dependence are also significantly related to obsessive compulsive symptoms.

Interpersonal sensitivity is significantly related to harm avoidance, self directedness and self transcendence. Self transcendence is no longer in the model after treatment. Depression is significantly related to self directedness and self transcendence at baseline and after treatment. Harm avoidance and reward dependence are also significantly related to depression after treatment. Harm avoidance, self directedness and self transcendence are significantly related to anxiety at both time points. After treatment, reward dependence and cooperativeness are also significantly related to anxiety.

Anger hostility is significantly related to all three character traits at baseline, after treatment anger hostility is only significantly related to self directedness. Harm avoidance, cooperativeness and self transcendence all significantly relate to phobic anxiety, however, after treatment harm avoidance and persistence are the significant variables in the model. The three character traits are significantly related to psychotocism at baseline. After treatment, cooperativeness is dropped and instead novelty seeking and reward dependence relate significantly to psychotocism, as well as the remaining two character traits.

The Males at Baseline

Table 5.5 presents the best GAM models for the males at baseline. As with the females, at both time points self directedness and self transcendence are important predictors of the symptom variables. However for the baseline males, reward dependence and persistence are also important. All the personality variables feature in at least one of the models.

The Males After Treatment

The post treatment models for the depressed males are presented in Table 5.6. Self directedness is still important as a predictor, cooperativeness now occurs in more models than self transcendence. Novelty seeking, harm avoidance and persistence all feature in at least one of the models, however, reward dependence is not in any of the models.

Comparison of Males Before and After Treatment

Somatisation is significantly related to cooperativeness and self transcendence at both time points. Self directedness is significantly related to obsessive compulsive at both time points. At baseline reward dependence and persistence are also significantly related to obsessive compulsive symptoms; after treatment this changes to just novelty seeking. Interpersonal sensitivity relates significantly to harm avoidance at both time points. At

Model Number	Symptom Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
MB 1	Somatisation	Cooperativeness	
		Self Transcendence	
MB 2	Obsessive Compulsive	Reward Dependence	
		Persistence	
		Self Directedness	
MB 3	Interpersonal Sensitivity	Harm Avoidance	Novelty Seeking
		Persistence	
		Self Directedness	
MB 4	Depression	Reward Dependence	
		Self Directedness	
MB 5	Anxiety	Reward Dependence	
		Self Directedness	
		Self Transcendence	
MB 6	Anger Hostility	Persistence	Self Transcendence
		Cooperativeness	
MB 7	Phobic Anxiety	Harm Avoidance	Novelty Seeking
		Self Transcendence	
MB 8	Paranoid Ideation	Persistence	Novelty Seeking
		Self Directedness	
		Cooperativeness	
		Self Transcendence	
MB 9	Psychotocism	Reward Dependence	
		Self Directedness	
		Cooperativeness	
		Self Transcendence	

Table 5.5: Best model from GAMS for the depressed males at baseline.

Model Number	Symptom Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
MP 1	Somatisation	Self Directedness	Cooperativeness
MP 2	Obsessive Compulsive	Novelty Seeking	
		Self Directedness	
MP 3	Interpersonal Sensitivity	Harm Avoidance	
		Cooperativeness	
MP 4	Depression	Novelty Seeking	
		Self Directedness	
MP 5	Anxiety	Harm Avoidance	Self Transcendence
		Persistence	
		Cooperativeness	
MP 6	Anger Hostility	Self Directedness	
		Self Transcendence	
MP 7	Phobic Anxiety	Harm Avoidance	Cooperativeness
			Persistence
MP 8	Paranoid Ideation	Cooperativeness	Self Transcendence
MP 9	Psychotocism	Self Directedness	
		Cooperativeness	

Table 5.6: Best model from GAMS for the depressed males post treatment.

baseline novelty seeking, persistence and self directedness are also in the model whereas after treatment cooperativeness is also in the model.

At baseline depression is significantly related to self directedness and reward dependence, after treatment reward dependence is no longer important and is replaced in the model by novelty seeking whilst self directedness remains. For anxiety, self transcendence is significantly related both before and after treatment. Before treatment reward dependence, and self directedness are also significantly related to anxiety. After treatment harm avoidance, persistence and cooperativeness are significantly related to anxiety, as well as self transcendence.

At baseline anger hostility relates significantly to persistence, cooperativeness and self transcendence. After treatment self transcendence is still significantly related to anger hostility, and self directedness is now in the model. Harm avoidance is common to the before and after models predicting phobic anxiety. Novelty seeking and self transcendence are important before treatment, and both cooperativeness and persistence are important after treatment.

At baseline paranoid ideation is significantly related to all three of the character traits as well as novelty seeking and persistence. After treatment only cooperativeness and self transcendence remain important. Psychotocism is related to all three character traits and reward dependence at baseline, after treatment only self directedness and cooperativeness

remain important.

Comparison of Males and Females

Comparing the baseline males to females the models have some similarities. All the models have some of the character traits common to both males and females. For example in the somatisation model, self transcendence occurs in both the males and females model. Only twice does a temperament trait occur in both the male and female model. Interpersonal sensitivity and phobic anxiety both have significant relationships with harm avoidance for both the males and females at baseline.

After treatment there are less common variables when comparing the male and female models. The character traits are still frequently common to both males and females. The temperament traits play more of a role with harm avoidance common across male and females in three of the models and persistence in one.

5.4.3 Investigation of the R^2

Table 5.7 presents the results from the investigation of the parametric (linear) models. All the F-tests are significant indicating the model is useful, however the R^2 and R^2_{adj} values are mostly very low. Four of the models have R^2_{adj} values that are above 30%. These models are all for the after treatment data and three are for the females (predicting obsessive compulsive, interpersonal sensitivity and anxiety) and one is for the males (predicting interpersonal sensitivity). These models are investigated further in section 5.4.4.

The R^2 and R^2_{adj} values for the nonparametric models are presented in Table 5.8. Seven of the models have R^2_{adj} values greater than 30%. In fact one model (predicting the males post treatment phobic anxiety) has an R^2_{adj} value greater than 50%. These models are presented in Section 5.4.4.

5.4.4 Investigation of the Models with $R^2_{adj} > 0.3$

The Female's Baseline Interpersonal Sensitivity

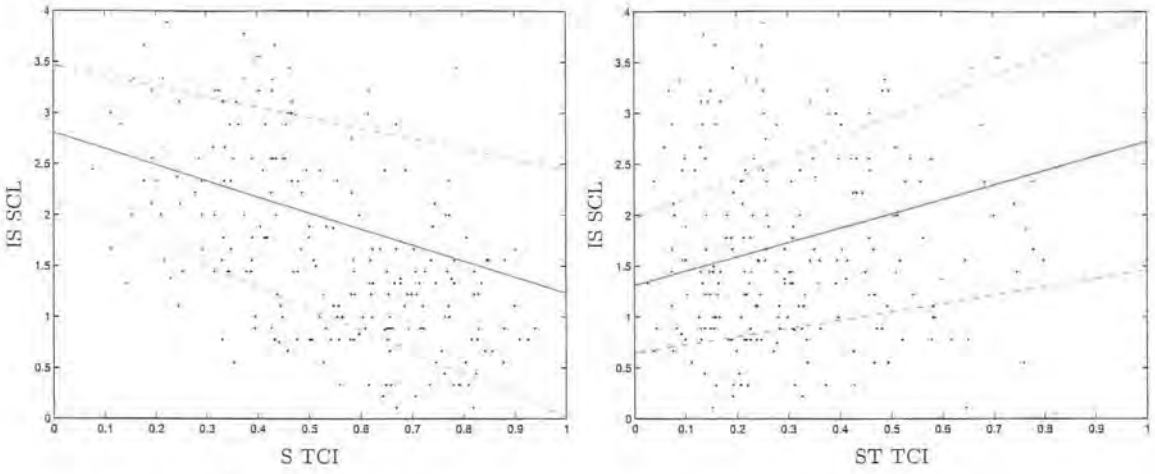
Only one model has an $R^2_{adj} > 0.3$ for the baseline females. This model predicts interpersonal sensitivity (Model FB9) and has an $R^2_{adj} = 0.3859$ This model has two linear predictors (self directedness and self transcendence) and one non-linear predictor (harm avoidance). Scatter plots were used to show the relationship between interpersonal sensitivity and the linear predictors (Figure 5.3). The regression lines with 95% confidence intervals are plotted as well. The regression line is calculated by holding all other variables constant. Self directedness (Figure 5.3(a)) has a negative relationship with interpersonal

Multiple Regression	Model Number	Models	F-test F	df_{num}	df_{den}	p-value	R^2	R^2_{adj}
Females Baseline	FB 1	F1 SCL	4.45	1	220	0.0361	0.0198	0.0154
	FB 2	F2 SCL	87.69	1	220	<0.0001	0.285	0.2817
	FB 3	F3 SCL	43.71	1	220	<0.0001	0.1657	0.1619
	FB 4	F4 SCL	21.15	1	220	<0.0001	0.0877	0.0836
	FB 6	F6 SCL	34.11	1	220	<0.0001	0.1342	0.1303
	FB 11	Anxiety	10.44	3	218	<0.0001	0.1256	0.1136
	FB 12	Anger Hostility	17.2	3	218	<0.0001	0.1914	0.1803
	FB 13	Phobic Anxiety	14.98	3	218	<0.0001	0.1709	0.1595
	FB 15	Psychotocism	27.88	3	218	<0.0001	0.2773	0.2674
Female Post Treatment	FP 1	F1 SCL	25.61	1	133	<0.0001	0.1615	0.1552
	FP 3	Somatisation	11.15	4	130	<0.0001	0.2555	0.2325
	FP 4	**Obsessive Compulsive**	15.59	4	130	<0.0001	0.3242	**0.3034**
	FP 5	**Interpersonal Sensitivity**	37.1	2	132	<0.0001	0.3598	**0.3501**
	FP 7	**Anxiety**	14.07	5	129	<0.0001	0.3529	**0.3278**
	FP 8	Anger Hostility	12.15	1	133	<0.0001	0.0837	0.0768
Males Baseline	MB 1	Somatisation	6.12	2	121	0.0029	0.0919	0.0769
	MB 2	Obsessive Compulsive	11.61	3	120	<0.0001	0.225	0.2056
	MB 4	Depression	9.93	2	121	0.0001	0.141	0.1268
	MB 5	Anxiety	10.45	3	120	<0.0001	0.2072	0.1874
	MB 9	Psychotocism	10.13	4	119	<0.0001	0.254	0.2289
Males Post treatment	MP 2	Obsessive Compulsive	13.43	2	64	<0.0001	0.2956	0.2735
	MP 3	**Interpersonal Sensitivity**	20.94	2	64	<0.0001	0.3955	**0.3766**
	MP 4	Depression	14.99	2	64	<0.0001	0.319	0.2977
	MP 6	Anger Hostility	4.86	2	64	0.0108	0.1319	0.1048
	MP 9	Psychotocism	12.37	2	64	<0.0001	0.2788	0.2563

Table 5.7: R^2 analysis of the multiple regression models.

Non-linear Models	Model Number		R^2	R^2_{adj}
Females Baseline	FB 5	F5 SCL	0.1611	0.1573
	FB 7	Somatisation	0.182	0.1707
	FB 8	Obsessive Compulsive	0.2123	0.2051
	FB 9	**Interpersonal Sensitivity**	0.3942	**0.3859**
	FB 10	Depression	0.2138	0.2066
	FB 14	Paranoid Ideation	0.2945	0.2815
Females Post Treatment	FP 2	F2 SCL	0.3015	0.2963
	FP 6	**Depression**	0.4165	**0.3985**
	FP 9	Phobic Anxiety	0.1693	0.1567
	FP 10	**Paranoid Ideation**	0.3716	**0.3523**
	FP 11	**Psychotocism**	0.4512	**0.4343**
Males Baseline	MB 3	Interpersonal Sensitivity	0.2804	0.2562
	MB 6	Anger Hostility	0.267	0.2487
	MB 7	Phobic Anxiety	0.2106	0.1909
	MB 8	Paranoid Ideation	0.3117	0.2825
Males Post Treatment	MP 1	Somatisation	0.2717	0.2489
	MP 5	**Anxiety**	0.4947	**0.4621**
	MP 7	**Phobic Anxiety**	0.5958	**0.5765**
	MP 8	**Paranoid Ideation**	0.3666	**0.3468**

Table 5.8: R^2 analysis of semi-parametric models.



(a) Self Directedness (b) Self Transcendence

Figure 5.3: Relationship between interpersonal sensitivity and the two linear predictor personality traits for the depressed females at baseline (Model 9, Table 5.3). Key: —— is the regression line , with – – – 95% confidence interval.

sensitivity whilst self transcendence (Figure 5.3(b)) has a positive relationship. Figure 5.4 presents the spline curve for the significant non-linear relationship between harm avoidance and interpersonal sensitivity.

The Female's Post Treatment Obsessive Compulsive

Obsessive compulsive symptoms were predicted linearly by four personality variables (Model FP 4, $R^2_{adj} = 0.3034$). Figure 5.5 presents scatter plots of the relationship between these four personality variables and obsessive compulsive symptoms. Harm avoidance and self transcendence are positively related to obsessive compulsive symptoms, whilst reward dependence and self directedness are negatively related. To better demonstrate the relationship two graphs were created that showed the temperament and character predictors with observations colour coded by quartile levels of obsessive compulsive. Figure 5.6(a) presents the temperament traits, harm avoidance versus reward dependence with the observations colour coded by quartile levels of obsessive compulsive. The higher values of obsessive compulsive symptoms are clustering around low reward dependence and high harm avoidance values. Figure 5.6(b) presents a similar graph with self directedness versus self transcendence and the points colour coded by obsessive compulsive. The graph shows that the lowest obsessive compulsive values (*) are clustering at high values of self directedness and low values of self transcendence.

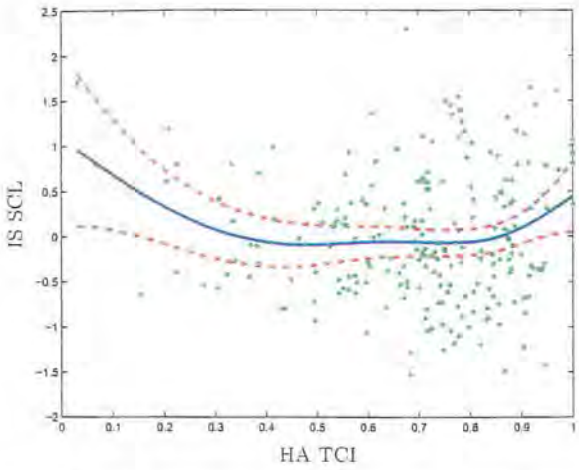


Figure 5.4: Spline curve for the partial prediction of interpersonal sensitivity by harm avoidance for the depressed females at baseline (Model 9, Table 5.3). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

The Female's Post Treatment Interpersonal Sensitivity

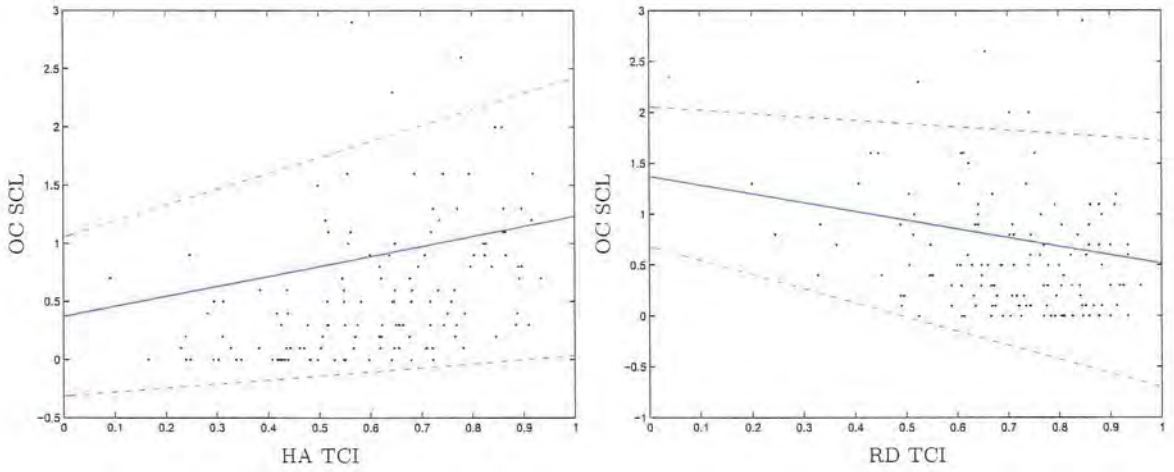
Harm avoidance and self directedness are linearly related to interpersonal sensitivity (Model FP 5, $R^2_{adj} = 0.3501$). The scatter plots in Figure 5.7 show that harm avoidance has a positive relationship and self directedness a negative one. The regression lines are plotted for each variable by holding all others constant.

Figure 5.8 presents harm avoidance versus self directedness with the points coded by interpersonal sensitivity. The observations below the lower quartile ($*$) are clustering towards the top left hand side of the graph where as the observations larger than the upper quartile ($\triangledown$) are clustering towards the bottom right hand corner. Patients with high values of harm avoidance and low values of self directedness tend to have high values of interpersonal sensitivity.

The Female's Post Treatment Depression

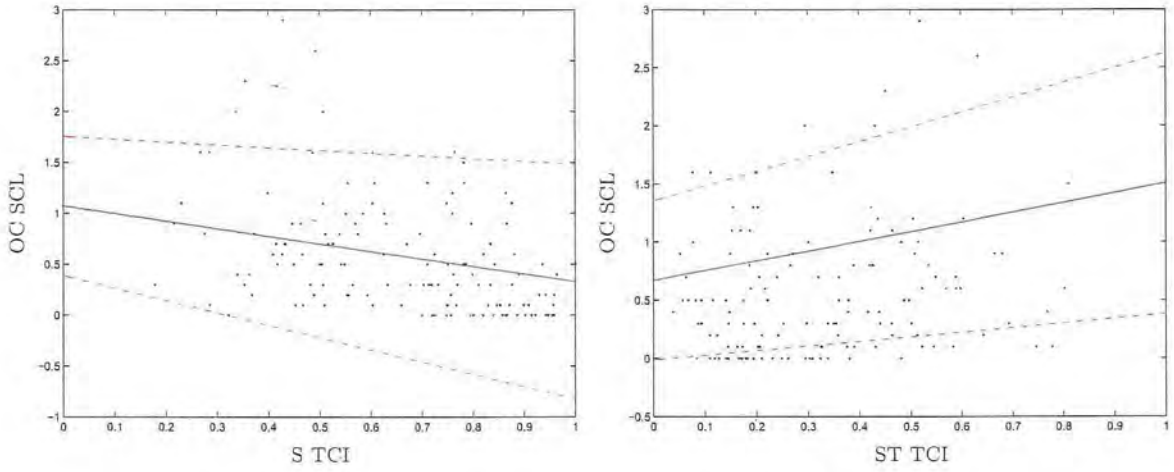
Depression (Model FP 6, $R^2_{adj} = 0.3985$) was linearly related to reward dependence, self directedness and self transcendence. It was also non-linearly related to harm avoidance. Reward dependence and self directedness both show negative relationships with depression whilst self transcendence has a positive relationship (Figure 5.9).

The non-linear relationship is shown in Figure 5.10. The lowest values of depression occur when harm avoidance is approximately 0.7. The values of depression increase away from this point. The residuals appear random.



(a) Harm Avoidance

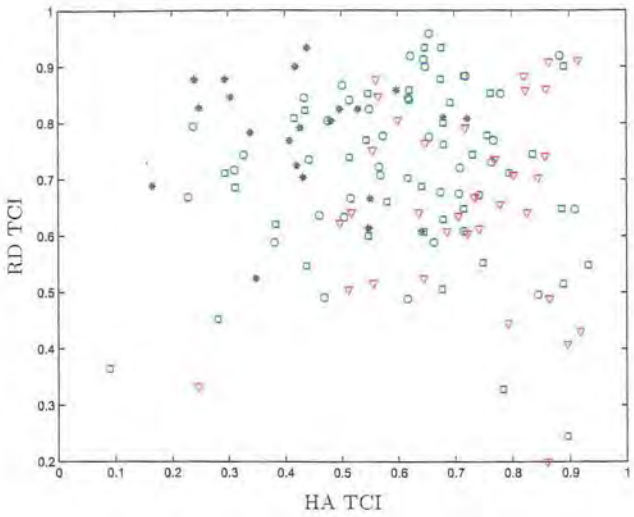
(b) Reward Dependence



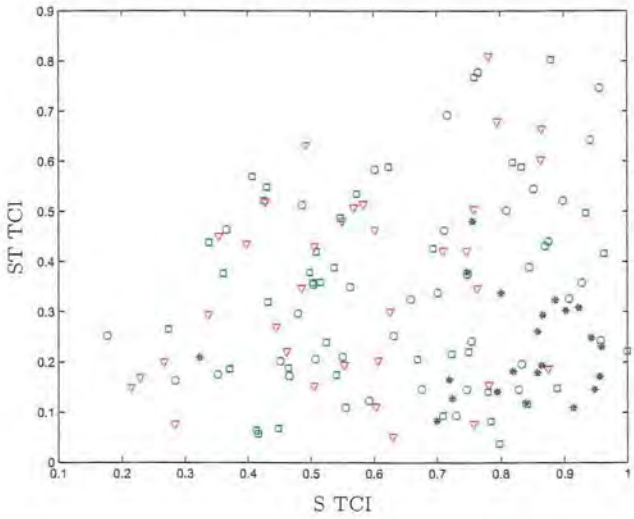
(c) Self Directedness

(d) Self Transcendence

Figure 5.5: Personality as predictors of obsessive compulsive symptoms for the depressed females after treatment (Model 4, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval.

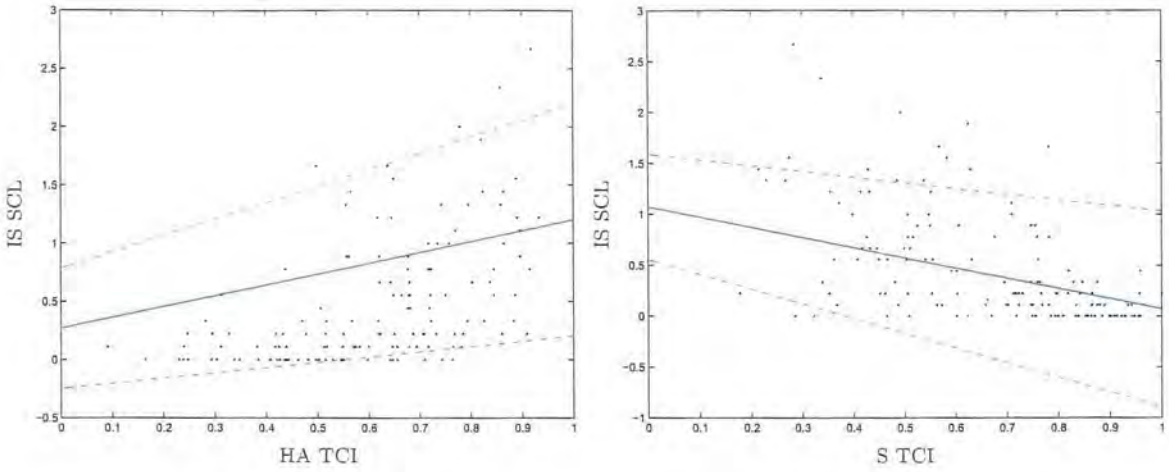


(a) Temperament Predictors



(b) Character Predictors

Figure 5.6: The four personality predictors of obsessive compulsive symptoms with the symptoms colour coded for the depressed females after treatment (Model 4, Table 5.4). Key: $*$ are the observations below Q1, $\circ$ are the observations between Q1 and Q2, $\square$ are the observations between Q2 and Q3, $\triangledown$ are the observations greater than Q3.



(a) Harm Avoidance (b) Self Directedness

Figure 5.7: Personality as predictors of interpersonal sensitivity for the depressed females after treatment (Model 5, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval.

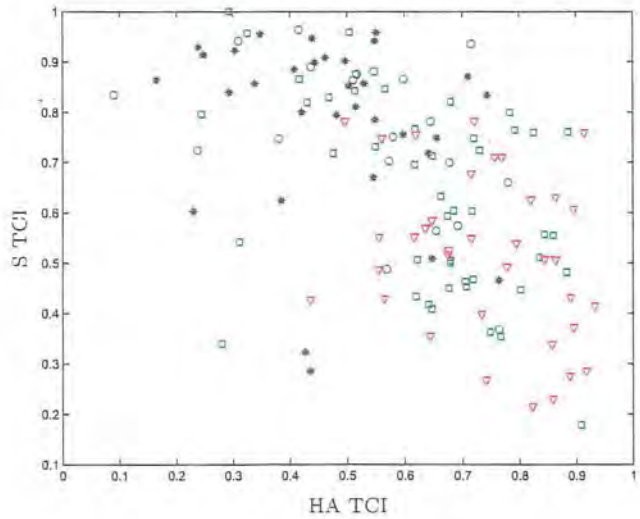
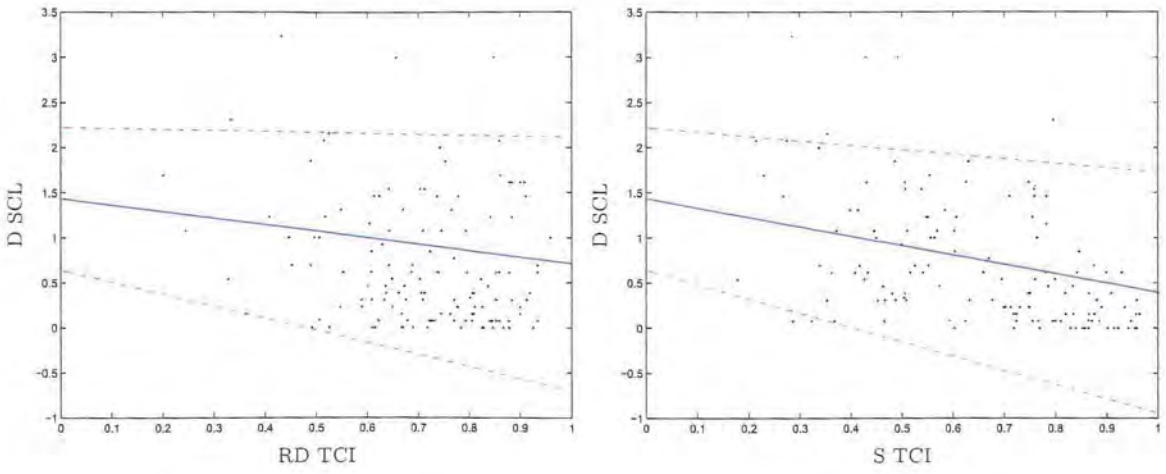
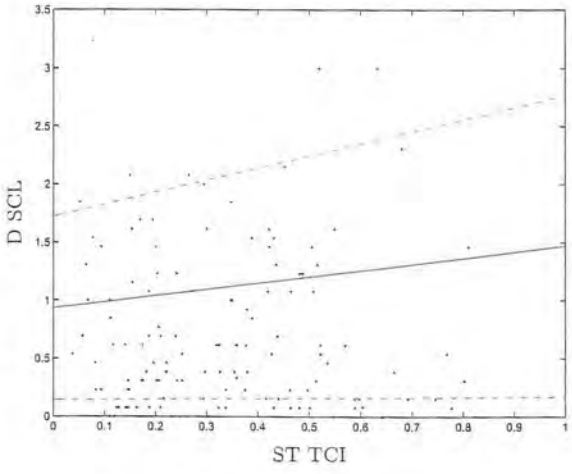


Figure 5.8: The two personality predictors of interpersonal sensitivity with the symptoms colour coded for the depressed females after treatment (Model 5, Table 5.4). Key: $*$ are the observations below Q1, $\circ$ are the observations between Q1 and Q2, $\square$ are the observations between Q2 and Q3, $\triangledown$ are the observations greater than Q3.



(a) Reward Dependence

(b) Self Directedness



(c) Self Transcendence

Figure 5.9: Relationship between depression and the three linear predictor personality traits for the depressed females after treatment (Model 6, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval.

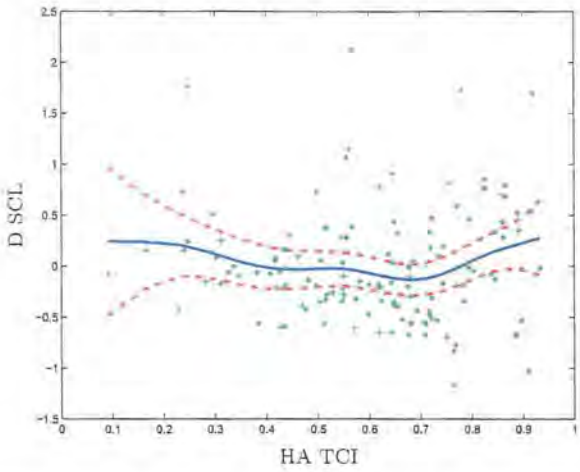


Figure 5.10: Spline curve for the partial prediction of depression by harm avoidance for the depressed females post treatment (Model 6, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

The Female's Post Treatment Anxiety

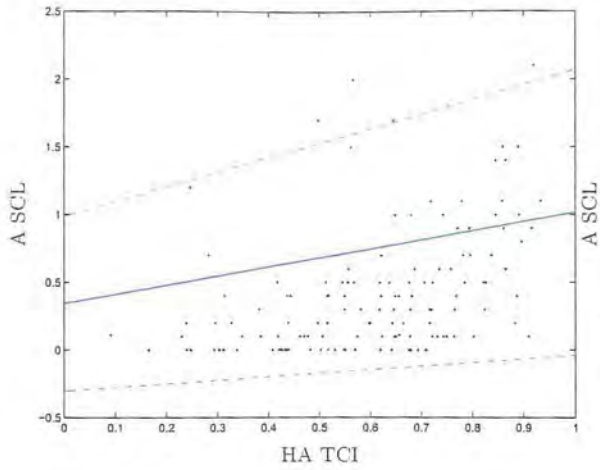
Increasing values of harm avoidance, cooperativeness and self transcendence, and decreasing values of reward dependence and self directedness, lead to increasing values of anxiety (Figure 5.11). About 33% of the variance in anxiety is described by the relationship with the five personality predictors (Model FP 7).

Figure 5.12(a) shows reward dependence versus harm avoidance, with quartile groupings of anxiety. Low values of anxiety (∗) occur for low values of harm avoidance and high values of reward dependence. The high values of anxiety (▽) tend to occur at high values of harm avoidance and low values of reward dependence. Figure 5.12(b) shows the character traits with observations coded by quartile levels of anxiety. Again there is separation between the low and high values of anxiety, across the three predictor variables.

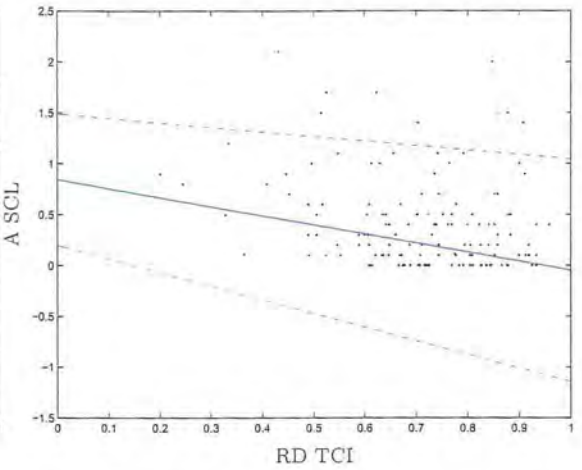
The Female's Post Treatment Paranoid Ideation

Paranoid ideation was related linearly to reward dependence, self directedness and self transcendence. It was also non-linearly related to cooperativeness (Model FP 10, $R^2_{adj} = 0.3523$). Figure 5.13 presents scatter plots for the three linear predictors. Reward dependence and self directedness show negative relationships and self transcendence a positive relationship.

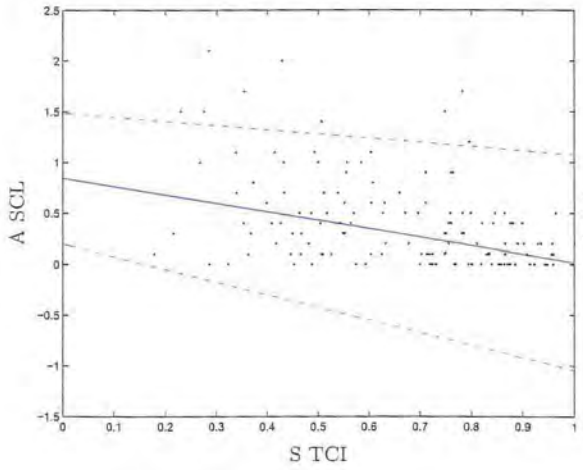
Figure 5.14 presents the spline curve for cooperativeness versus paranoid ideation. The highest paranoid ideation values occur for low cooperativeness values, decreasing from there to a minimum at around 0.6 (for cooperativeness). The paranoid ideation values slowly increase from that point.



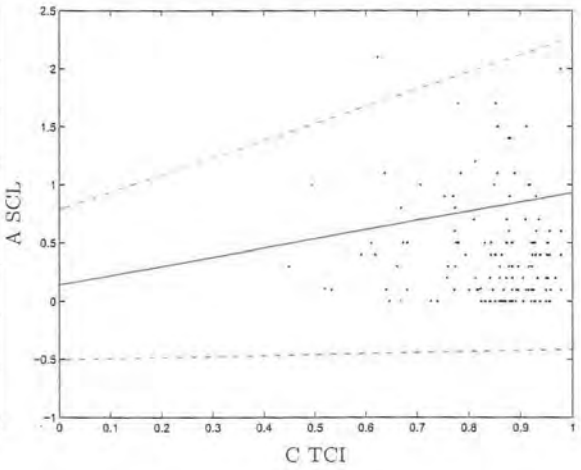
(a) Harm Avoidance



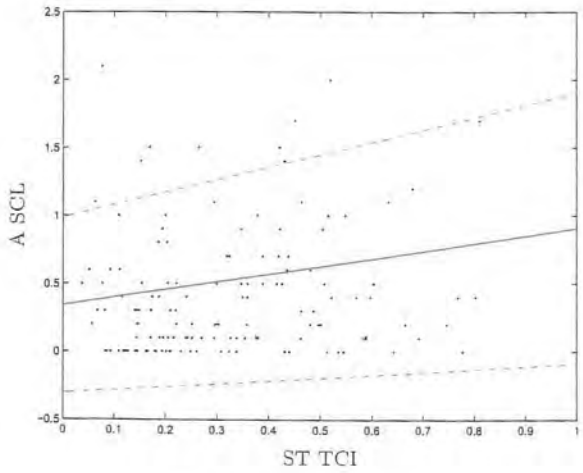
(b) Reward Dependence



(c) Self Directedness

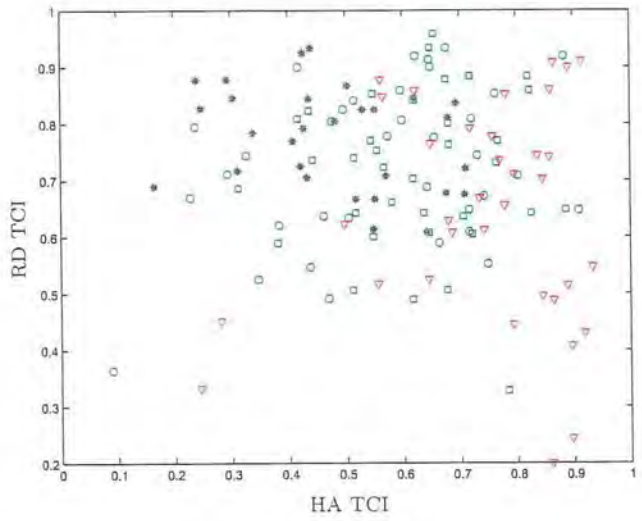


(d) Cooperativeness

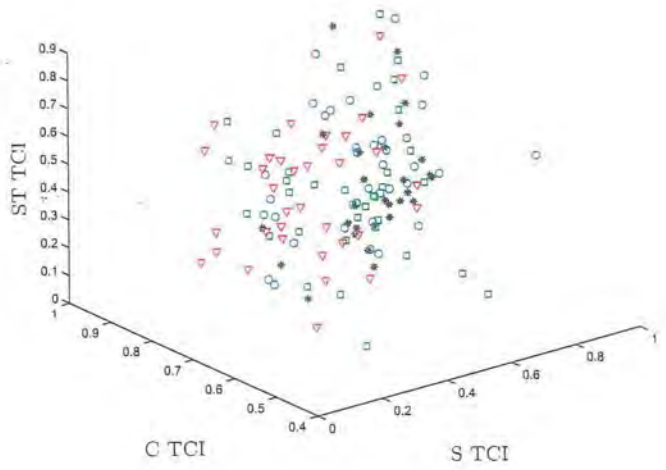


(e) Self Transcendence

Figure 5.11: Personality as predictors of anxiety for the depressed females after treatment (Model 7, Table 5.4). Key: —— is the regression line , with - - - 95% confidence interval.

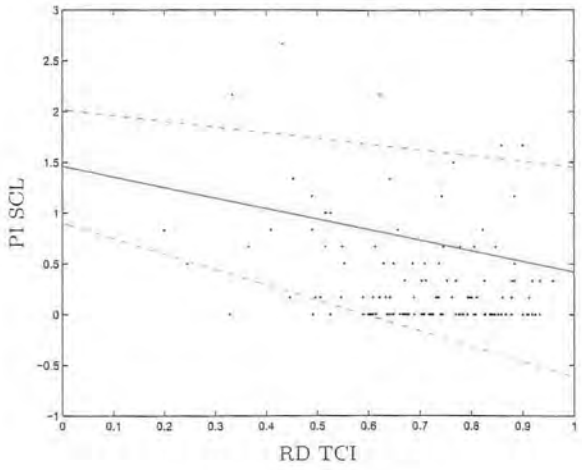


(a) Temperament Predictors

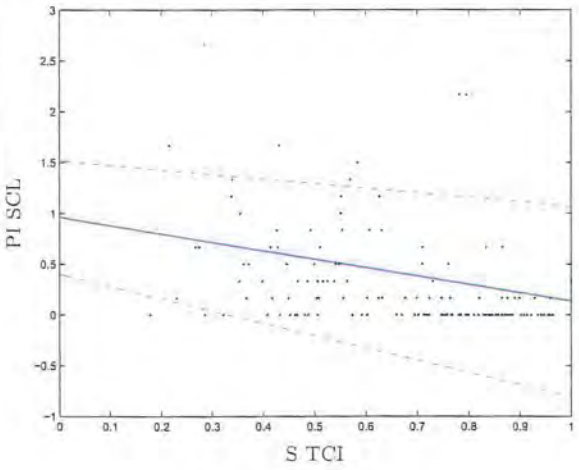


(b) Character Predictors

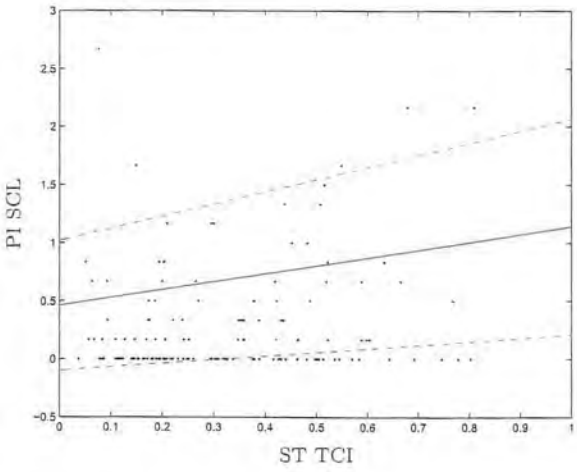
Figure 5.12: The five personality predictors of anxiety with the symptoms colour coded for the depressed females after treatment (Model 7, Table 5.4). Key: $*$ are the observations below Q1, $\circ$ are the observations between Q1 and Q2, $\square$ are the observations between Q2 and Q3, $\triangledown$ are the observations greater than Q3.



(a) Reward Dependence



(b) Self Directedness



(c) Self Transcendence

Figure 5.13: Relationship between paranoid ideation and the three linear predictor personality traits for the depressed females after treatment (Model 10, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval.

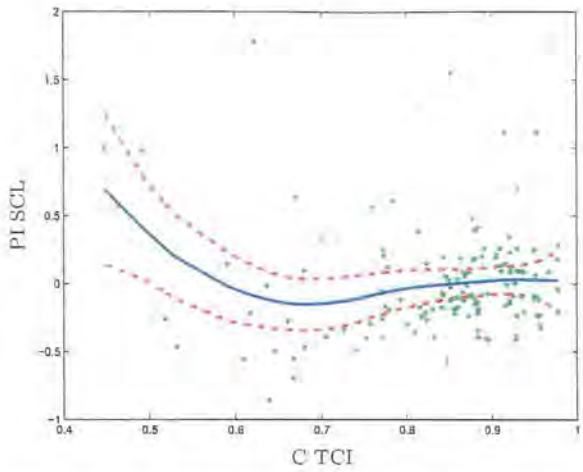


Figure 5.14: Spline curve for the partial prediction of paranoid ideation by cooperativeness for the depressed females post treatment (Model 10, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

The Female's Post Treatment Psychotocism

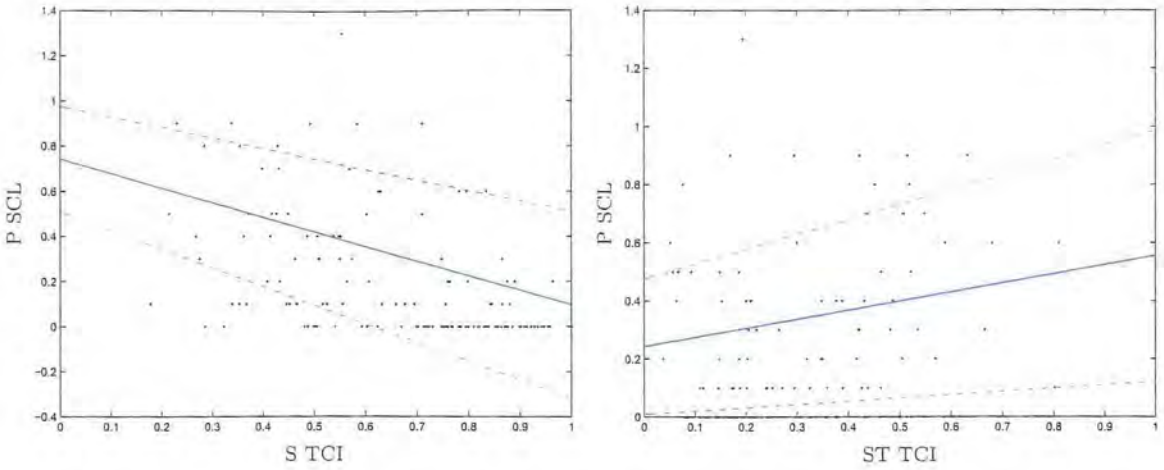
The model predicting psychotocism (Model FP 11, $R^2_{adj} = 0.4343$) for the depressed females after treatment involves novelty seeking, reward dependence, self directedness and self transcendence. Self directedness has a negative linear relationship with psychotocism (Figure 5.15(a)). Self transcendence has a positive relationship with psychotocism (Figure 5.15(b)). Novelty seeking and reward dependence have non-linear relationships with psychotocism (Figure 5.16). These spline curves show the shape of the non-linearity.

The Baseline Males

The depressed males at baseline have personality variables that are poor predictors of the symptoms. All the best models found had R^2_{adj} values below 30%. The highest value was 28% for the model predicting paranoid ideation. Due to the low R^2_{adj} values none of the baseline models were investigated further.

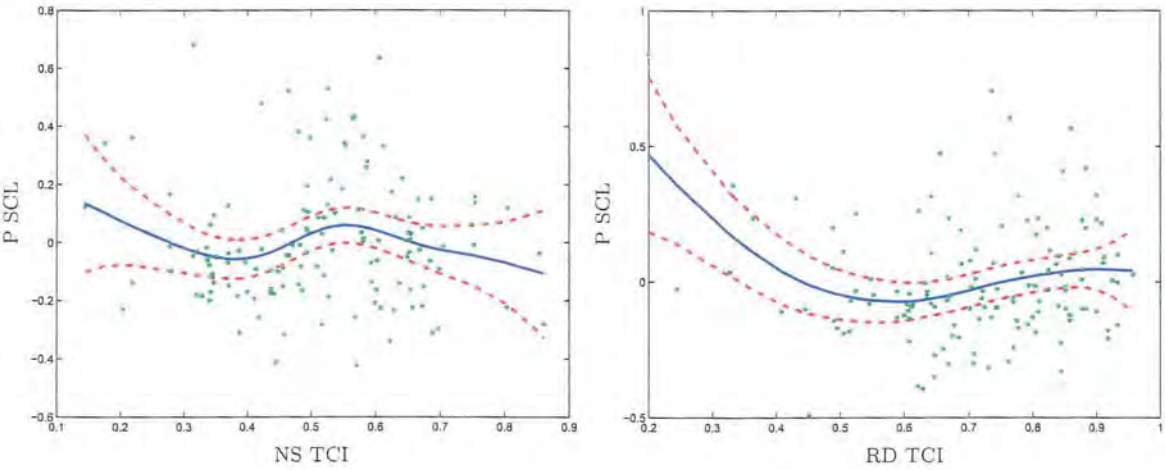
The Male's Post Treatment Interpersonal Sensitivity

Increasing harm avoidance levels and decreasing cooperativeness levels are related to increasing interpersonal sensitivity levels (Figure 5.17, Model MP 3, $R^2_{adj} = 0.3766$). This is further demonstrated in Figure 5.18 where harm avoidance is plotted versus cooperativeness and the observations are coded by interpersonal sensitivity. Higher values (□ and ▽) of interpersonal sensitivity are at the right side and lower values (∗ and ○) are towards the left side of the graph.



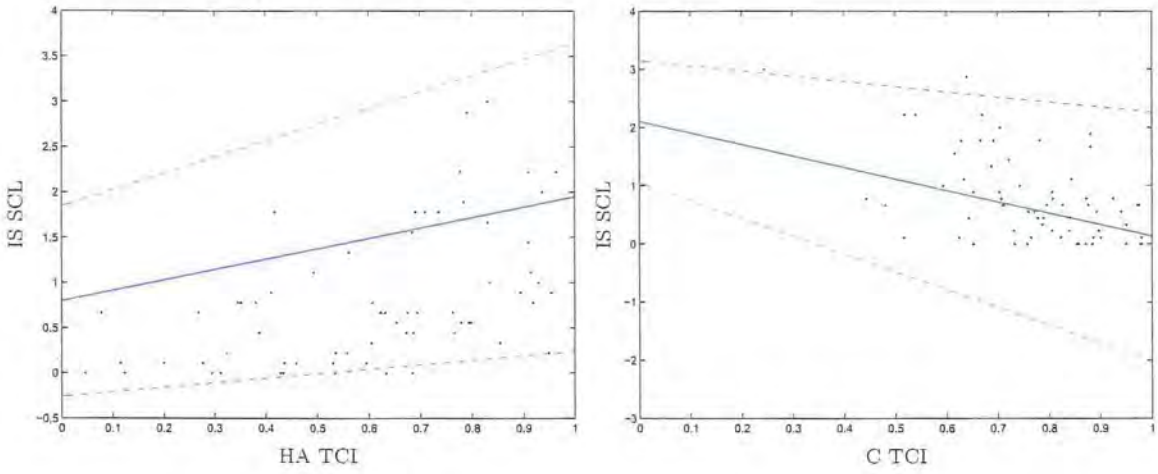
(a) Self Directedness (b) Self Transcendence

Figure 5.15: Relationship between psychotocism and the two linear predictor personality traits for the depressed females after treatment (Model 11, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval.



(a) Novelty Seeking (b) Reward Dependence

Figure 5.16: Spline curves for the partial prediction of psychotocism for the depressed females post treatment (Model 11, Table 5.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.



(a) Harm Avoidance (b) Cooperativeness

Figure 5.17: Personality as predictors of interpersonal sensitivity for the depressed males after treatment (Model 3, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval.

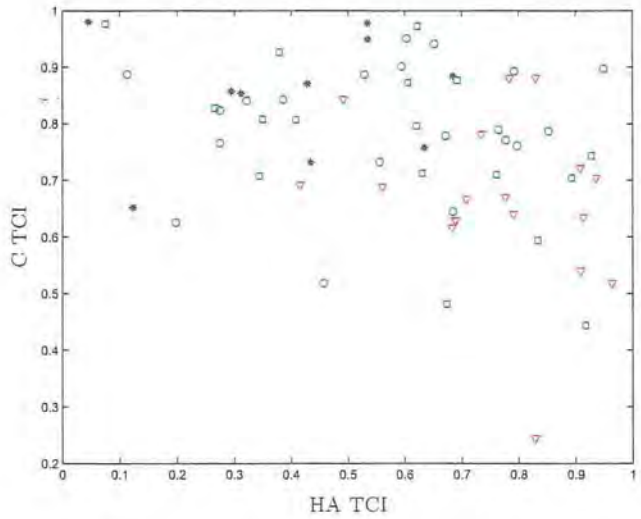


Figure 5.18: The two personality predictors of interpersonal sensitivity with the symptoms colour coded for the depressed males after treatment (Model 3, Table 5.6). Key: $*$ are the observations below Q1, $\circ$ are the observations between Q1 and Q2, $\square$ are the observations between Q2 and Q3, $\triangledown$ are the observations greater than Q3.

The Male's Post Treatment Anxiety

Anxiety (Model MP 5, $R^2_{adj} = 0.4621$) has three linear predictors, harm avoidance, persistence and cooperativeness. Self transcendence is the fourth predictor and has a non-linear relationship with anxiety. Higher values of harm avoidance and persistence and lower values of cooperativeness tend to give higher values of anxiety (Figure 5.19). The non-linear relationship is presented in Figure 5.20. Anxiety values are increasing for self transcendence values from 0.1 to 0.3 and 0.6 to 0.9. Anxiety values decrease from about 0.3 to 0.6 on the self transcendence scale.

The Male's Post Treatment Phobic Anxiety

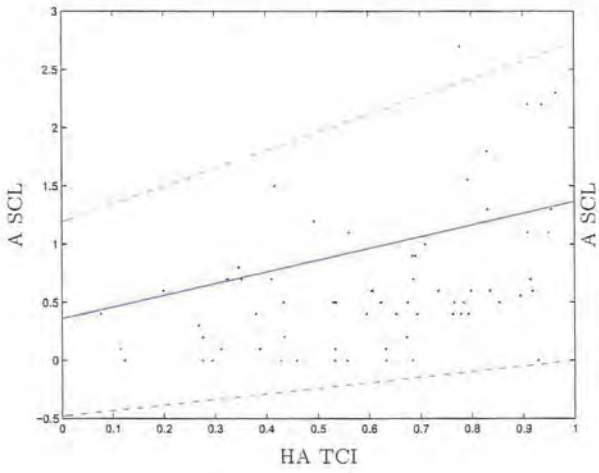
Increasing harm avoidance is related to increasing phobic anxiety (Figure 5.21, Model MP 7, $R^2_{adj} = 0.5765$). Persistence and cooperativeness have non-linear relationships with phobic anxiety (Figure 5.22). The highest phobic anxiety scores occur for a value of persistence around 0.5 and for low values of cooperativeness. About 57% of the variance in phobic anxiety is explained by the relationship with harm avoidance, persistence and cooperativeness. There appears to be an extreme value at low cooperativeness values that may be having an effect on the model. The model was recalculated with this point removed and the resulting spline curves are presented in Figure 5.23. The spline curve for persistence, Figure 5.23(a), looks similar to that before removal (Figure 5.22(a)), however it is no longer significantly non-linear. Likewise the curve for cooperativeness (Figure 5.23(b)) is not significantly non-linear but there is a significant linear relationship between cooperativeness and phobic anxiety at the 5% significance level. The variance in phobic anxiety accounted for the model was recalculated giving $R^2 = 0.4368$ and $R^2_{adj} = 0.4095$. There has been a substantial drop in these values by removing the outlier.

The Male's Post Treatment Paranoid Ideation

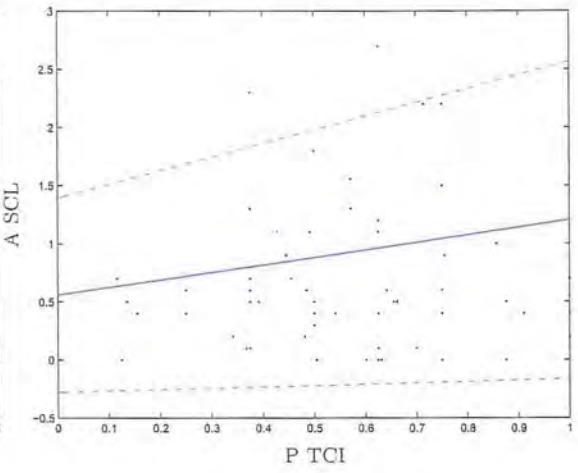
Cooperativeness is a negative linear predictor of paranoid ideation (Figure 5.24, Model MP 8, $R^2_{adj} = 0.3468$). Self transcendence has a non-linear relationship with paranoid ideation. Essentially paranoid ideation values decrease as self transcendence increases to a value of about 0.55, then paranoid ideation decreases as self transcendence increases.

5.4.5 Overview

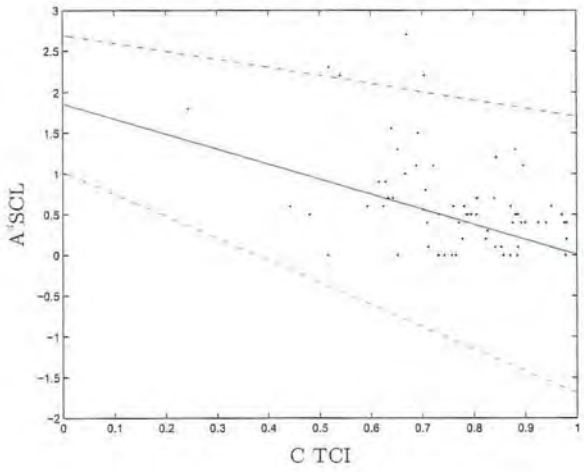
The models show that significant relationships exist between personality and symptoms, however the personality variables are generally poor predictors of symptoms. The path analysis models, analysed in Section 5.4.1, showed poor fit and insignificant loadings for



(a) Harm Avoidance



(b) Persistence



(c) Cooperativeness

Figure 5.19: Relationship between anxiety and the three linear predictor personality traits for the depressed males after treatment (Model 5, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval.

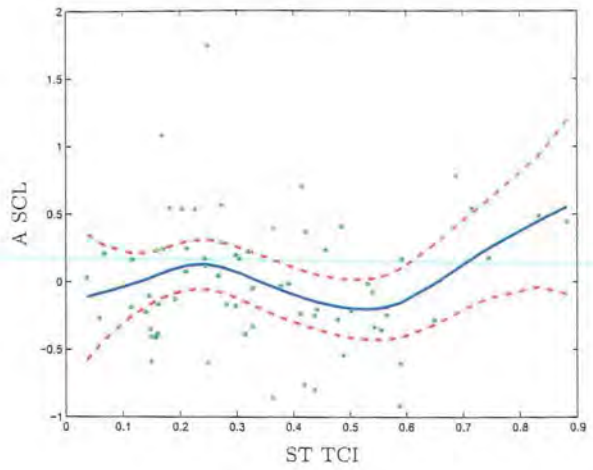


Figure 5.20: Spline curve for the partial prediction of anxiety by self transcendence for the depressed males post treatment (Model 5, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

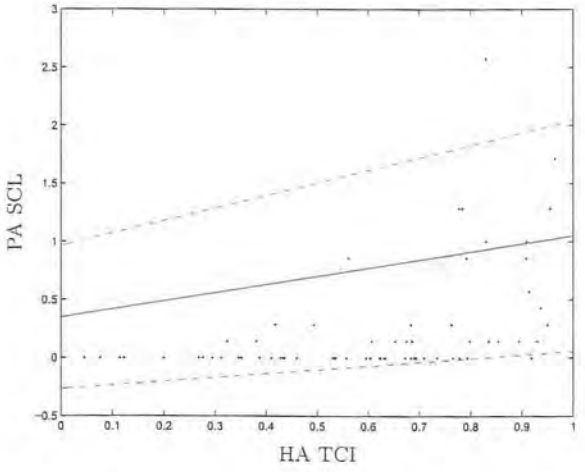
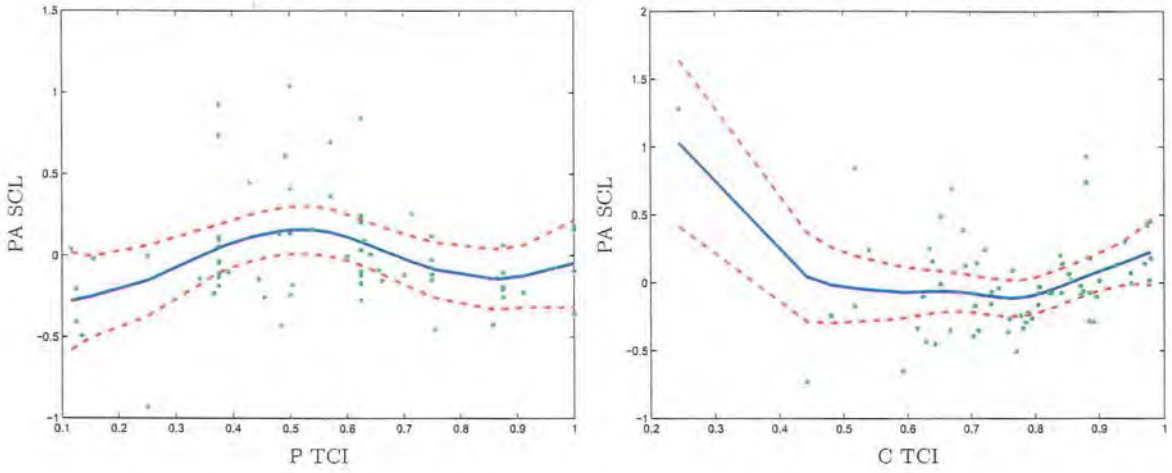
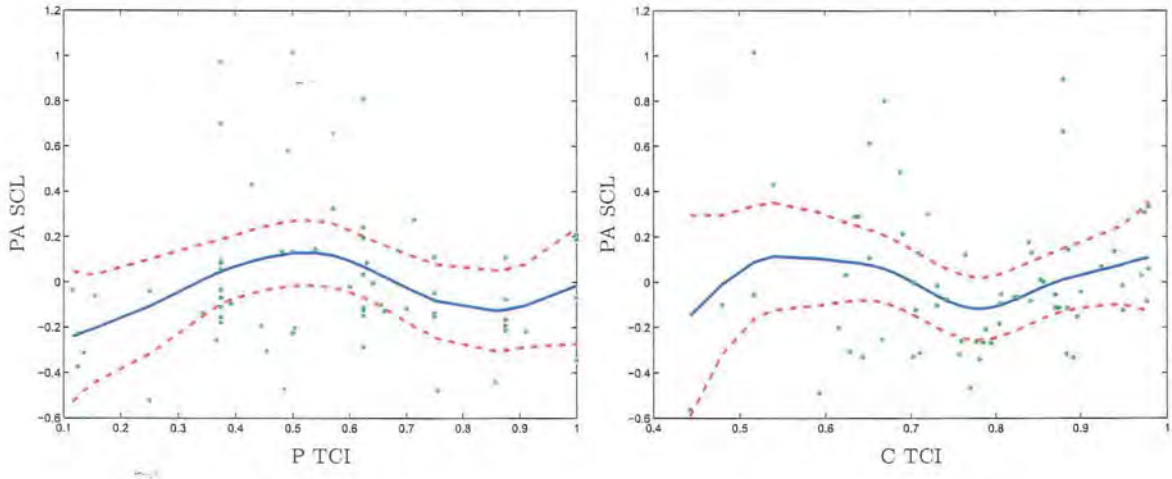


Figure 5.21: Relationship between phobic anxiety and the linear predictor harm avoidance for the depressed males after treatment (Model 7, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval.



(a) Persistence (b) Cooperativeness

Figure 5.22: Spline curves for the partial prediction of phobic anxiety for the depressed males post treatment (Model 7, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.



(a) Persistence (b) Cooperativeness

Figure 5.23: Spline curves after removal of potential outliers for the partial prediction of phobic anxiety for the depressed males post treatment (Model 7, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

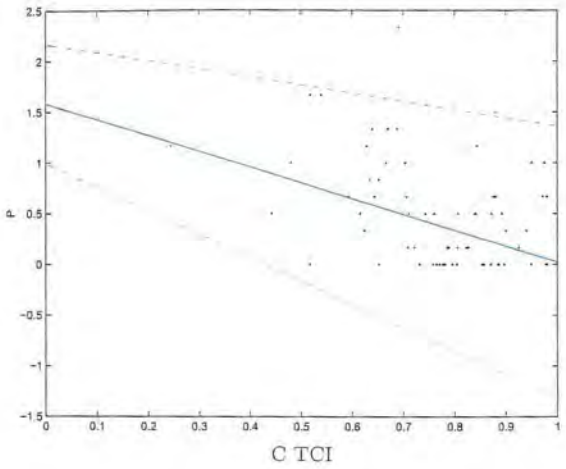


Figure 5.24: Relationship between paranoid ideation and the linear predictor cooperativeness for the depressed males after treatment (Model 8, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval.

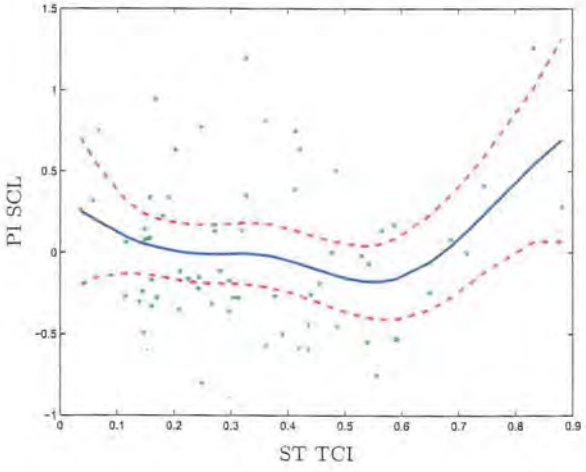


Figure 5.25: Spline curve for the partial prediction of paranoid ideation by self transcendence for the depressed males post treatment (Model 8, Table 5.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

the regression terms from the personality factor to the symptom factors. These results are similar to those found in Section C.1 where the directionality was the other way around.

The GAM models found from the model building steps were presented in Section 5.4.2 and further analysed using the R^2 and R^2_{adj} statistics in Section 5.4.3. There were a number of significant linear and non-linear relationships. Eleven models had an $R^2_{adj} > 0.3$. The model with the highest R^2_{adj} was the prediction of phobic anxiety by harm avoidance, cooperativeness and persistence for the depressed males after treatment (Model MP 7, Table 5.8). This model had an R^2_{adj} value of 0.58. Thus the model explains 58% of the variance in phobic anxiety. However, after removal of an extreme value the R^2_{adj} dropped to 41%.

The Females at Baseline

The baseline models are presented below. Only one model (in bold) had an R^2_{adj} value greater than 30%. In general the character traits (self directedness, cooperativeness and self transcendence) are the important variables for relating personality to symptoms. The temperament trait harm avoidance features in a number of the models.

$$F1_{SCL} = 0.11F1_{TCI} + 0.00$$

$$F2_{SCL} = 0.49F1_{TCI} + 0.00$$

$$F3_{SCL} = 0.24F1_{TCI} + 2.23$$

$$F4_{SCL} = 0.17F1_{TCI} + 0.65$$

$$F5_{SCL} = f(F1_{TCI})$$

$$F6_{SCL} = 0.22F1_{TCI} + 1.13$$

$$S_{SCL} = 0.95HA_{TCI} + 1.27ST_{TCI} + f(NS_{TCI}) - 0.06$$

$$OC_{SCL} = 1.20ST_{TCI} + f(S_{TCI}) + 1.95$$

$$\mathbf{IS_{SCL} = -1.57S_{TCI}+0.67ST_{TCI}+f(HA_{TCI})+1.31}$$

$$D_{SCL} = 0.62ST_{TCI} + f(S_{TCI}) + 2.90$$

$$A_{SCL} = 0.55HA_{TCI} - 0.70S_{TCI} + 0.90ST_{TCI} + 1.10$$

$$AH_{SCL} = -1.01S_{TCI} - 1.68C_{TCI} + 0.93ST_{TCI} + 2.72$$

$$PA_{SCL} = 1.36HA_{TCI} - 1.01C_{TCI} + 0.88ST_{TCI} + 0.22$$

$$PI_{SCL} = -1.77C_{TCI} + 0.94ST_{TCI} + f(HA_{TCI}) + f(S_{TCI}) + 2.42$$

$$P_{SCL} = -1.26S_{TCI} - 0.64C_{TCI} + 0.76ST_{TCI} + 1.85$$

The Males at Baseline

The models for the males at baseline are presented below. None of the models had an R^2_{adj} value more than 30%. Whilst significant relationships exist as shown below, the personality variables are poor predictors of symptoms. As with the females at baseline, the character traits are important in all of the models, though there are more relationships between the temperament traits and the symptoms than the females showed.

$$S_{SCL} = -0.89C_{TCI} + 0.82ST_{TCI} + 1.36$$

$$OC_{SCL} = 0.88RD_{TCI} + 0.61P_{TCI} - 1.70S_{TCI} + 1.95$$

$$IS_{SCL} = 1.09HA_{TCI} + 0.67P_{TCI} - 0.86S_{TCI} + f(NS_{TCI}) + 0.71$$

$$D_{SCL} = 0.79RD_{TCI} - 1.32S_{TCI} + 2.37$$

$$A_{SCL} = 1.18RD_{TCI} - 1.16S_{TCI} + 0.78ST_{TCI} + 1.07$$

$$AH_{SCL} = 0.61P_{TCI} - 1.94C_{TCI} + f(ST_{TCI}) + 2.21$$

$$PA_{SCL} = 1.00HA_{TCI} + 0.74ST_{TCI} + f(NS_{TCI}) - 0.60$$

$$PI_{SCL} = 0.74P_{TCI} - 0.96S_{TCI} - 1.02C_{TCI} + 1.06ST_{TCI} + f(NS_{TCI}) + 1.52$$

$$P_{SCL} = 0.64RD_{TCI} - 0.75S_{TCI} - 1.17C_{TCI} + 0.87ST_{TCI} + 1.64$$

The Females after Treatment

The female after treatment models are presented below. Six of the models had R^2_{adj} values greater than 30%, these models are shown in bold. The character traits are still important in the relationship between personality and symptoms, harm avoidance and reward dependence are also important. This suggests that, after treatment, the temperament traits contribute to the relationship more so than at baseline.

$$F1_{SCL} = 0.10F1_{TCI}$$

$$F2_{SCL} = f(F1_{TCI})$$

$$S_{SCL} = 0.65HA_{TCI} - 0.78RD_{TCI} - 0.34P_{TCI} + 0.48ST_{TCI} + 0.56$$

$$\mathbf{OC_{SCL} = 0.86HA_{TCI} - 0.86RD_{TCI} - 0.74S_{TCI} + 0.83ST_{TCI} + 0.87}$$

$$\mathbf{IS_{SCL} = 0.93HA_{TCI} - 1.00S_{TCI} + 0.57}$$

$$\mathbf{D_{SCL} = -0.72RD_{TCI} - 1.04S_{TCI} + 0.54ST_{TCI} + f(HA_{TCI}) + 0.93}$$

$$\mathbf{A_{SCL} = 0.67HA_{TCI} - 0.89RD_{TCI} - 0.83S_{TCI} + 0.79C_{TCI} + 0.56ST_{TCI} + 0.34}$$

$$AH_{SCL} = -0.65S_{TCI} + 0.74$$

$$PA_{SCL} = 0.57HA_{TCI} + f(P_{TCI}) - 0.13$$

$$\mathbf{PI_{SCL} = -1.04RD_{TCI} - 0.83S_{TCI} + 0.68ST_{TCI} + f(C_{TCI}) + 1.96}$$

$$\mathbf{P_{SCL} = -0.65S_{TCI} + 0.31ST_{TCI} + f(NS_{TCI}) + f(RD_{TCI}) + 0.94}$$

The Males after Treatment

The males post treatment models are presented below. Four of the models had an R^2_{adj} value greater than 30%, shown in bold. The model for the prediction of phobic anxiety had the highest R^2_{adj} value of all the models presented in this chapter with a value of 58% before removal of an extreme value. As with the females, the temperament traits are much more important in the post treatment models than the baseline models, though the character traits still have significant contributions.

$$S_{SCL} = -0.78S_{TCI} + f(C_{TCI}) + 1.20$$

$$OC_{SCL} = -1.25NS_{TCI} - 1.68S_{TCI} + 2.49$$

$$\mathbf{IS_{SCL} = 1.15HA_{TCI} - 1.96C_{TCI} + 1.59}$$

$$D_{SCL} = -1.07NS_{TCI} - 1.89S_{TCI} + 2.63$$

$$\mathbf{A_{SCL} = 1.01HA_{TCI} + 0.64P_{TCI} - 1.85C_{TCI} + f(ST_{TCI}) + 0.86}$$

$$AH_{SCL} = -0.78S_{TCI} + 0.69ST_{TCI} + 0.67$$

$$\mathbf{PA_{SCL}} = \mathbf{0.70HA_{TCI} + f(C_{TCI}) + f(P_{TCI}) + 0.85}$$

$$\mathbf{PI_{SCL}} = \mathbf{-1.56C_{TCI} + f(ST_{TCI}) + 1.38}$$

$$P_{SCL} = -0.64S_{TCI} - 0.90C_{TCI} + 1.46$$

Comparison to Original Hypotheses

The first hypothesis was that depressed patients with a high total symptom score would have high harm avoidance and low self directedness. Whilst a total score was not used for this study, all the symptoms are positively correlated, as evidenced by the positive loadings on the structural models, so looking across all the symptoms, an indication of total symptom severity can be seen. Indeed, particularly at baseline at least one of self directedness or harm avoidance occurs in all except two of the baseline models. Harm avoidance has a positive relationship and self-directedness has a negative relationship. In two models harm avoidance or self directedness have non-linear relationships.

The second hypothesis related high anxiety to high harm avoidance. This relationship can be seen in the female's baseline anxiety model and in both the males and females post treatment anxiety models. High novelty seeking and low cooperativeness are hypothesised to relate to high anger hostility. At baseline low cooperativeness is related to high anger hostility, however novelty seeking does not feature in the model. After treatment this hypothesised relationship is not found.

High obsessive compulsive scores were hypothesised to be related to low novelty seeking, high harm avoidance, low reward dependence and high persistence. At baseline for the females obsessive compulsive model, none of the temperament traits were found in the model. The males at baseline had a relationship between high reward dependence and persistence, and high obsessive compulsive. After treatment the females had a relationship between high harm avoidance, low reward dependence and high obsessive compulsive values. The males, after treatment, had a relationship between low novelty seeking and high obsessive compulsive. Some of the original hypothesised relationships are seen but more so after treatment.

The last hypothesis suggested a gender specific link between high somatisation and high novelty seeking, harm avoidance, reward dependence and persistence. At baseline, the females had a significant positive relationship between harm avoidance and somati-

sation. The male's baseline model only contains character traits. After treatment, the female's somatisation scores are related to high harm avoidance, low reward dependence and low persistence. The male's model still only uses character traits.

Predicting the Depression Symptom from Personality

At baseline, depression is related to self transcendence and self directedness for the females, and reward dependence and self directedness for the males. Both models have an $R^2_{adj} < 0.3$ so whilst a significant relationship exists the personality variables are poor predictors of the symptom variable. A similar pattern is seen for the males after treatment, this time novelty seeking and self-directedness are significantly related to depression. Again the R^2_{adj} is less than 0.30 so the personality variables are poor predictors of the males post treatment depression. The females after treatment have a model that accounts for 40% of the variance in the depression symptom. In the model depression is related to low reward dependence and self directedness, high self transcendence and a non-linear function of harm avoidance. It is important to note that self-directedness is significantly related to depression before and after treatment for both males and females.

Interpreting the Best Models

At baseline 39% of the variance of the females interpersonal sensitivity is accounted for by the model (Table 5.8). From the model low self-directedness, high self-transcendence and a non-linear function of harm avoidance predict high interpersonal sensitivity. The non-linear function (Figure 5.4) shows the interpersonal sensitivity values decrease for the low harm avoidance scores, stay similar for mid-range harm avoidance scores and increase for the very high harm avoidance scores. The males had no models at baseline that accounted for more than 30% of the variance in the predicted symptom variable.

After treatment six of the eleven female models have an $R^2_{adj} > 0.3$. The first model relates high obsessive compulsive to high harm avoidance, low reward dependence, low self-directedness and high self-transcendence. The second relates high interpersonal sensitivity to high harm avoidance and low self directedness. The third model predicts depression linearly from reward dependence, self-directedness and self-transcendence and non-linearly from harm avoidance. The non-linear relationship shown in Figure 5.10 has the lowest depression scores when harm avoidance has a score of approximately 0.7. Away from this point depression scores increase.

Anxiety has five linear personality predictors. High harm avoidance, cooperativeness and self transcendence, and low reward dependence and self-directedness related to high anxiety values. The fifth model relates paranoid ideation linearly to negative reward dependence and self-directedness, positive self-transcendence and non-linearly to coop-

erativeness. From Figure 5.14, the non-linear relationship shows high paranoid ideation values for low cooperativeness with paranoid ideation values decreasing to cooperativeness values of about 0.7. Away from this point depression scores increase.

The final model for the post treatment females to have an $R^2_{adj} > 0.3$ is the prediction of psychotocism by self directedness, self transcendence, novelty seeking and reward dependence. Self directedness has a negative linear relationship with psychotocism whilst self transcendence has a positive linear relationship. Both novelty seeking and reward dependence have significant non-linear relationships with psychotocism. The spline curves were presented in Figure 5.16. Psychotocism scores decrease for novelty seeking values from 0.1 to 0.35 and 0.55 to 0.9. Psychotocism scores increase over the range of novelty seeking values from 0.35 to 0.55. Psychotocism scores decrease for increasing reward dependence values up to a value of about 0.6. From then on psychotocism scores increase slowly.

For the post treatment males five of the nine models have an $R^2_{adj} > 0.3$. The first model predicts interpersonal sensitivity from harm avoidance and cooperativeness. Harm avoidance has a positive linear relationship with interpersonal sensitivity and cooperativeness has a negative linear relationship. In the second model high harm avoidance and persistence values, and low cooperativeness values as well as the non-linear contribution from self-transcendence predict high values of anxiety. The non-linear relationship, shown in Figure 5.20, shows that as self-transcendence increases from 0 to 0.25 and 0.55 to 0.9, anxiety values also increase. Anxiety values decrease for self-transcendence values in the range 0.25 to 0.55.

Phobic anxiety is modelled linearly by harm avoidance and non-linearly by cooperativeness and persistence. This model had the highest R^2_{adj} value of 0.58. Harm avoidance had a positive linear relationship with phobic anxiety. Figure 5.22 shows the non-linear relationships. Generally persistence values of about 0.5 had the highest phobic anxiety values. Phobic anxiety values decreased away from this point except for the persistence values in the range 0.9 to 1. Phobic anxiety decreases rapidly as cooperativeness increases to a value of about 0.45, from that point the decrease is slower. After cooperativeness values of 0.8 phobic anxiety values increase. An observation can be seen at the low cooperativeness end that is well away from most of the data. The analyses were redone with this point removed. This had a dramactic effect on the model dropping the R^2_{adj} to 41% compared to the original 58%.

The final model predicts paranoid ideation from cooperativeness and self transcendence. Low cooperativeness values predict high paranoid ideation values. Paranoid ideation values decrease over self transcendence values ranging from 0 to approximately 0.55. From this value on paranoid ideation values increase.

5.5 Summary

The studies to date have shown that personality has some importance in predicting the outcomes of depressed patients. The studies using the Cloninger model have concentrated on the temperament traits, only recently has the literature shown more studies investigating all seven of the Cloninger traits. Harm avoidance and self directedness consistently feature in study results. The work presented in this chapter has thoroughly investigated the relationship between symptoms of depression and all seven of the Cloninger personality traits both before and after treatment. This chapter used the methods of path analysis and general additive models. Again, these methods do not appear to have been used previously in this area. This study also has a larger sample size than the majority of those studies and has not only used personality as a predictor of symptoms but also symptoms as a predictor of personality.

The first important result is the importance that the character traits played in most of the models particularly at baseline. The temperament traits were more important after treatment than they had been at baseline. The personality variables appeared to be better predictors of symptoms than the other way around as in the first instance eleven models had an R^2_{adj} greater than 30% and only three models had an R^2_{adj} greater than 30% for the prediction of personality from symptoms. Many of the symptoms are reduced to zero upon improvement therefore the interesting question is which levels of personality traits lead to the low post treatment scores. The models after treatment tended to have a higher R^2_{adj} than at baseline. One of the eleven models was for the baseline data.

The study shows that general additive modelling is of value for this type of data and allows for a more thorough investigation of potential relationships. The path analysis showed very poor fit and this could be due to a number of reasons. A likely explanation is the path analysis only models linear relationships and models these relationship simultaneously.

The study also has the advantage over the literature of investigating male and females separately. Different models were found for the males and females with some similarity of variables across the models. Further areas for the study to investigate would be to use the baseline personality as a predictor of a measure of the improvement in symptoms and to investigate if the success of the different depression treatments can be related to the personality types.

Chapter 6

Brain Function Analysis

Recently developed non-invasive methods for investigating brain function have allowed functional brain information, rather than structural information, to be related to psychological measures. These methods have largely been applied to individuals with disorders such as schizophrenia (Curtis et al., 2001; Ebmeier et al., 1993), and mood disorders, including depression (Meyer et al., 2001; Videbech et al., 2001; Mayberg et al., 1999; Bench et al., 1995). Less attention has been paid to investigating correlations between brain function and personality traits within normal subjects, using these non-invasive methods.

The non-invasive methods developed, in the last thirty years, to investigate brain function, as distinct from brain structure, include single photon emission computed tomography (SPECT) (Prohovnik, 1993; Drevous, 1989), positron emission tomography (PET) (Ter-Pogossian, 1985) and functional magnetic resonance imaging (fMRI) (Callicott et al., 1998). This study uses SPECT to measure brain function. SPECT images contain counts of received radiation in each pixel (or voxel). The count is in proportion to the blood flow at the time of tracer injection, giving an indicator of oxygen uptake, and thus brain function.

While it has been accepted that mental disorder such as schizophrenia, bipolar disorder and depression arise from abnormalities of neurotransmitter function in specific brain regions, it is becoming accepted that 'normal emotional experiences', (Mayberg et al., 1999) and 'normal personality traits' will also be related to changes in neurotransmitter function in specific regions. For instance, the personality trait of 'detachment', which represents individuals' description of themselves as cold and socially aloof, is related to dopamine D2 receptor density (Breier et al., 1998; Farde et al., 1997). One specific model of personality, developed by Cloninger, was explicitly based upon an association of specific personality traits to an underlying neurobiology (Cloninger, 1986). One of the temperament dimensions in the model, reward dependence, is strongly negatively correlated to the trait of 'detachment' described above (Breier et al., 1998).

Personality profiles within normal subjects are starting to be investigated using functional imaging techniques. A recent study (Johnson et al., 1999) related regional cerebral blood flow (rCBF) to the NEO personality inventory (Costa and McCrae, 1985) and showed the introversion/extroversion personality dimension to be significantly related to specific brain regions. Sugiura et al. (2000) were the first to investigate the Temperament and Character Index in relation to rCBF. They found significant relationships between rCBF and the three investigated temperament traits (novelty seeking, harm avoidance and reward dependence).

During the 1980's analysis of functional brain images used "regions of interest" to relate covariates to blood flow. This method involved predetermining particular brain regions in which the average blood flow for the region was related to some covariate. The regions were manually pre-specified leading to bias of only investigating areas that were thought to be associated with the covariate, potentially leaving out areas that related to the covariate. This methodology restricted analysis to larger pre chosen areas. These problems motivated the development of voxel-by-voxel analysis techniques (Friston et al., 1990, 1991) that are currently in vogue. These techniques model blood flow at each and every voxel, and then each voxel is checked for a significant relationship whilst taking into account the highly dependent nature of the voxels. The first such method was developed by Friston et al. (1990, 1991).

This chapter uses the recently developed technique of statistical parametric mapping (SPM) (Friston et al., 1995a,c, 1994; Frackowiak et al., 1997) to investigate the relationship between regional cerebral blood flow and personality types, in the 20 normal males aged between 20 and 33 years (see Chapter 2). Quartile personality variables were developed and used as a predictor of blood flow. The resulting models were analysed using t-contrasts. The work furthers that of Sugiura et al. (2000) by investigating all seven of Cloninger's TCI personality traits. The results have been published in the Journal of NeuroImage (Turner et al., 2003).

6.1 Overview of Statistical Parametric Mapping

There are three main procedures involved with statistical parametric mapping. The first step is the stereotactic normalisation and smoothing discussed in Sections 6.2 and 6.3. This shifts and warps the brains into a standard brain space so that analysis of a particular voxel is an investigation of the same three dimensional piece in all the subjects. Smoothing conditions the data for the statistical analysis. The second step is the development and calculation of the general linear model (GLM) to be analysed at each and every voxel across the subjects (Section 6.4.1). The GLM models the blood flow in each voxel as dependent on a combination of conditions, covariates or nuisance variables. The final

step is the analysis of the model at each and every voxel for significant effects, whilst taking into account the high spatial dependence inherent to brain images. This process is discussed in 6.4.2.

6.2 Image Preprocessing

The images obtained from SPECT (see Section 1.5.1 for the details of the SPECT reconstruction process), or indeed any other brain imaging modality, are not in a standard space. Whilst a standard head position was aimed for during scanning, this cannot be achieved without a very accurate alignment system. Even so, each brain is in a slightly different position in the skull and is different in size across subjects. Non-label based image transformation methods (Friston et al., 1995a) are the best way of aligning all the brain images into a standard space. Spatial normalisation and realignment, implemented within the SPM99 (Welcome Department of Cognitive Neurology, London, UK, www.fil.ion.ucl.ac.uk/spm, Friston et al. (1995a,c, 1994)) software, was used to transform the brain images into the standard MNI space (Evans et al., 1993). MNI space is a standard brain space developed from 305 MRI volumes by the Montreal Neurological Institute.

During early brain imaging studies, images were aligned using label based methods. Particular landmarks in the brain were identified then the brains aligned so that the landmarks coincided. This method relied on the landmarking abilities of the user. To counter this user bias automatic non label based methods were developed. The following section presents the theory for the nonlabel based stereotactic normalisation used within SPM99. The following theory was introduced and expanded by Friston (1995); Friston et al. (1995a); Frackowiak et al. (1997).

If the images were from the same person a rigid body transformation could be used to align the brains. The affine transformation can be used to achieve this. The first step involves finding the optimal affine (rigid body) transformation then secondly smooth deformations are applied. The affine transformation maps the points (x_0, y_0, z_0) to the coordinates of some second space or image. A rigid body transformation will translate and rotate the image to the same orientation as the template. For a translation in x, y and z by a, b and c and a rotation about the x, y and z axes by Θ, Φ and Ω (radians) the following matrices are needed

$$\text{translation} \quad \begin{pmatrix} 1 & 0 & 0 & a \\ 0 & 1 & 0 & b \\ 0 & 0 & 1 & c \\ 0 & 0 & 0 & 1 \end{pmatrix} \tag{6.1}$$

$$x\text{-axis rotation} \quad \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & \cos\Theta & \sin\Theta & 0 \\ 0 & -\sin\Theta & \cos\Theta & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix} \tag{6.2}$$

$$y\text{-axis rotation} \quad \begin{pmatrix} \cos\Phi & 0 & \sin\Phi & 0 \\ 0 & 1 & 0 & 0 \\ -\sin\Phi & 0 & \cos\Phi & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix} \tag{6.3}$$

$$z\text{-axis rotation} \quad \begin{pmatrix} \cos\Omega & \sin\Omega & 0 & 0 \\ -\sin\Omega & \cos\Omega & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}. \tag{6.4}$$

If the images are from different subjects, not only does the image need to be translated and rotated, but also deformed. This is the case when normalising the subjects to the template image. This is achieved in the following way, using the theory from Friston (1995).

The difference between two images $(Y(\mathbf{x}), Z(\mathbf{x}))$ can be separated into two components. The first an intensity transform (f_x) and the second a spatial discrepancy (q) giving

$$f_x(Y(\mathbf{x})) = Z(q(\mathbf{x})) + e(\mathbf{x}) \tag{6.5}$$

for some scalar function of error $e(\mathbf{x})$. To solve the equation, and thus perform the transformation, an explicit least squares solution is sought for both f_x and q by linearisation. The linearisation uses low order approximations and constraints. The constraints chosen give smoothness to the image and preserve local contiguity relationships.

Let f_x be a function $\gamma_x(Y(\mathbf{x}))$ convolved with a kernel c as in equation 6.6.

$$f_x(Y(\mathbf{x})) = c * \gamma_x(Y(\mathbf{x})), \tag{6.6}$$

where $*$ is a convolution. Expand $\gamma_x(\ )$ as $\gamma_x(Y(\mathbf{x})) = \sum u_i(\mathbf{x}) f_i(\mathbf{x})$ when $u_i(\mathbf{x})$ are the position dependent coefficients which can be rewritten in terms of smooth basis functions $\beta_j(\mathbf{x})$. Substituting these into equation 6.5 gives,

$$c * \sum [f_i(Y(\mathbf{x})) \sum u_{ij} \beta_j(\mathbf{x})] \approx Z(\mathbf{x} + \sum q_k \beta_k(\mathbf{x})), \tag{6.7}$$

and using a first order Taylor series approximation gives

$$Z(\mathbf{x} + \sum q_k \beta_k(\mathbf{x})) \approx Z(\mathbf{x}) + \sum q_k \frac{\partial Z(\mathbf{x})}{\partial q_k}, \tag{6.8}$$

$$\frac{\partial Z(q(\mathbf{x}))}{\partial q_k} = \beta_k(\mathbf{x}).\nabla Z(\mathbf{x}). \tag{6.9}$$

Combining these results leads to

$$\sum\sum u_{ij}[c * f_i(Y(\mathbf{x})).\beta_j(\mathbf{x})] \approx Z(\mathbf{x}) + \sum q_k \frac{\partial Z(\mathbf{x})}{\partial q_k}. \tag{6.10}$$

The left hand side of equation 6.10 shows that the transformation can be found by convolving the intensity transform. The right hand side of the equation shows the approximate distortions, which are the additive effects of each component of the distortion, which are defined by a set of smooth basis functions. Equation 6.10 is linear in u_{ij} and q_k, which are the unknown coefficients.

To find the least squares solution of equation 6.10 we rewrite it in matrix form giving

$$[\mathbf{c}.diag(f_0(Y)).\beta \quad \mathbf{c}.diag(f_1(Y)).\beta \ \ldots \ -\frac{\partial Z}{\partial q}].[\mathbf{u_0}\ \mathbf{u_1}\ \ldots\ \mathbf{q}]^T \approx Z \tag{6.11}$$

for a Toeplitz matrix $\mathbf{c}$ of the convolution kernel c, with $Y(\,)$ and $Z(\,)$ column vectors of the image voxels. Let $\mathbf{A} = [\mathbf{c}.diag(f_0(Y)).\beta \quad \mathbf{c}.diag(f_1(Y)).\beta \ \ldots \ -\frac{\partial Z}{\partial q}]$ then equation 6.11 becomes

$$\mathbf{A}.[\mathbf{u_0}\ \mathbf{u_1}\ \ldots\ \mathbf{q}]^T \approx Z, \tag{6.12}$$

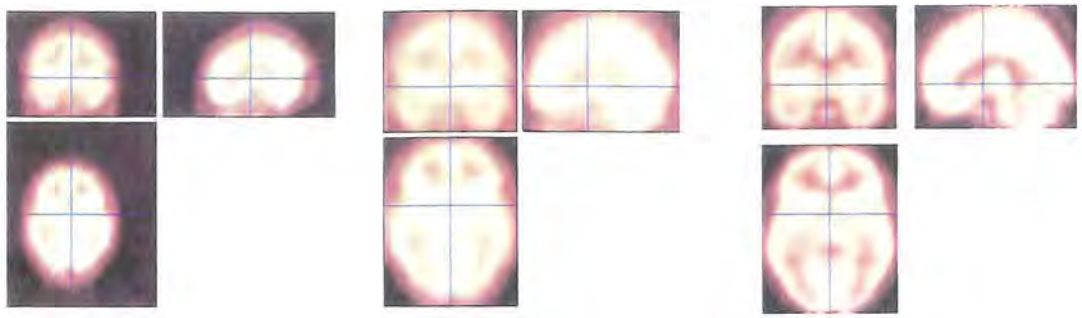
and

$$[\mathbf{u_0}\ \mathbf{u_1}\ \ldots\ \mathbf{q}]^T \approx (\mathbf{A}^T.\mathbf{A})^{-1}\mathbf{A}^T Z \tag{6.13}$$

giving the least squares solution for the unknown coefficients. A further simplification can be made, as the resolution of the images is the same within modality (all the images are from SPECT), using a first order approximation on γ_x to give

$$[diag(Y).\beta \quad -\frac{\partial Z}{\partial \mathbf{q}}].[\mathbf{u_1} \quad \mathbf{q}]^T \approx Z. \tag{6.14}$$

The above process works better for images closer together, so the images were manually readjusted closer to the template prior to the stereotactic normalisation. Figure 6.1 presents a raw brain image, the same image after stereotactic normalisation and the template image. Ignoring the scale differences from the separate presentation of each image, we can see there has been a reshaping of the brain to match the template.



(a) Raw Image (b) Normalised Image (c) SPECT Template

Figure 6.1: Comparison of raw and normalised image with template.

(a) 15mm Smoothing (b) 20mm Smoothing (c) 25mm Smoothing

Figure 6.2: Comparison of smoothing levels.

6.3 Smoothing

The images were smoothed, with a Gaussian filter, to condition the data for the statistical analyses. The amount of smoothing was determined by some preliminary tests of the statistical analyses to varying smoothing amounts. The optimum smoothing was found to be of size 20mm FWHM (full width at half maximum) in all three dimensions. Figure 6.2 presents some preliminary result clusters, under different smoothing conditions. There is definite improvement from 15mm to 20mm smoothing. There appears to be a slight improvement from 20mm to 25mm smoothing, but across a number of models, 20mm smoothing was the optimum.

6.4 Statistical Parametric Mapping

Statistical Parametric Mapping developed due to the need to analyse functional brain images that had a large number of responses for few stimuli, a situation difficult to analyse using standard statistical techniques. The technique, as implemented in SPM99, uses the general linear model at each and every voxel simultaneously to construct a map characterising the null hypothesis at each voxel. This map is assessed for significant voxels or regions (see Section 6.4.2).

6.4.1 The General Linear Model

The general linear model is a parametric method for assessing the null hypothesis, in this case, of no relationship between the covariate and the blood flow in the particular voxel. The model has L explanatory variables (x_{jl}) which elicit a response variable (Y_j) for observations $j = 1, \ldots, J$. There must be more observations than explanatory variables ($L < J$). This is not the case when treating the brain voxels together as the response variables, so each voxel is modelled individually (i.e. predicted from personality). Multiple comparison testing, across the 500000+, are taken into account while assessing the significance of the model parameters. Following the notation and theory used in Frackowiak et al. (1997) the general linear model is formulated as follows

$$Y_j = x_{j1}\beta_1 + \cdots + x_{jl}\beta_l + \cdots + x_{jL}\beta_L + \epsilon_j, \tag{6.15}$$

ϵ_j are error terms which are assumed to be independent and identically distributed with zero mean and variance σ^2. The β's are the unknown coefficients. The matrix formulation of the general linear model is as follows

$$\begin{pmatrix} Y_1 \\ \vdots \\ Y_j \\ \vdots \\ Y_J \end{pmatrix} = \begin{pmatrix} x_{11} & \ldots & x_{1l} & \ldots & x_{1L} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{j1} & \ldots & x_{jl} & \ldots & x_{jL} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{J1} & \ldots & x_{Jl} & \ldots & x_{JL} \end{pmatrix} \begin{pmatrix} \beta_1 \\ \vdots \\ \beta_j \\ \vdots \\ \beta_J \end{pmatrix} + \begin{pmatrix} \epsilon_1 \\ \vdots \\ \epsilon_j \\ \vdots \\ \epsilon_J \end{pmatrix}. \tag{6.16}$$

The matrix of explanatory variables (x_{jl}) is known as the design matrix. Each column represents one model parameter and each row represents one observation. Least squares estimation is usually used to solve the system, which in most cases is unique. This model is developed simultaneously at each voxel retaining the same design matrix but allowing the parameters to vary. Further details can be found in Friston et al. (1995c).

In this study personality was modelled univariately as a predictor of blood flow at each voxel using the SPM99 software. The images were globally normalised using proportional

scaling to stabilise the variance (Frackowiak et al., 1997), with total blood flow scaled to 50ml/min/dl. The following equations (Frackowiak et al., 1997) were used for this calculation:

$$y_j^k = y_j^k/(g_j/50)$$

where y_j^k is the blood flow, for scan j, at voxel $k = 1, \ldots, K$ ($K = 510340$ for this study), and g_j is the global flow of scan j calculated by:

$$g_j = (\sum_{k=1}^{K} y_j^k)/K$$

Initial regression models using the personality scores as predictors of blood flow did not show a significant linear relationship between blood flow and personality. The results presented in this chapter show that the relationship is non-linear, hence the inability for the initial models to detect a relationship. Grouping the personality data into quartiles (25^{th}, 50^{th} and 75^{th} percentiles) has often been more informative and is an approach that has been used in personality studies (e.g. Fergusson et al. (2003)). The personality groupings (Q1 - Q4), as predictors of blood flow in each voxel, can be written as a general linear model of the following form:

$$y_j^k = \beta_{1j}^k x_{1j}^k + \beta_{2j}^k x_{2j}^k + \beta_{3j}^k x_{3j}^k + \beta_{4j}^k x_{4j}^k + \epsilon_j^k$$

where

$$x_{qj}^k = \delta_{qQ} \qquad \delta_{qQ} = \begin{cases} 1 & q = Q \\ 0 & \text{otherwise} \end{cases}$$

$k = 1, \ldots, K$ voxels — $j = 1, \ldots, 20$ subjects

$q = 1, \ldots, 4$ — β = coefficients

ϵ_j^k = error term — Q = four quartile groups

Seven models were tested, using the seven traits (grouped into quartiles) individually as predictors of regional cerebral blood flow (rCBF).

Least squares estimation is used to solve the above equation to get estimates of the coefficients ($\widehat{\beta}$). Least squares estimates minimise the residual sum of squares, defined as

$$S = \sum_{j=1}^{J} (Y_j - x_{j1}\widehat{\beta}_1 - \cdots - x_{j4}\widehat{\beta}_4)^2. \tag{6.17}$$

To minimise equation 6.17 differentiate and set equal to zero as follows

$$\frac{\partial S}{\partial \widehat{\beta}_i} = 2\sum_{j=1}^{J} (-x_{ji})(Y_j - x_{j1}\widehat{\beta}_1 - \cdots - x_{j4}\widehat{\beta}_4) = 0, \tag{6.18}$$

or rewritten in matrix notation

$$X^T\mathbf{Y} = (X^TX)\widehat{\boldsymbol{\beta}}. \tag{6.19}$$

For a design matrix X that is full rank (X^TX) and invertible, equation 6.19 can be solved for the least squares estimates of the β coefficients as follows,

$$\widehat{\boldsymbol{\beta}} = (X^TX)^{-1}X^T\mathbf{Y}. \tag{6.20}$$

These $\widehat{\boldsymbol{\beta}}$ are maximum likelihood estimates assuming normally distributed errors.

The F-test can be used for measuring the overall adequacy of the model, across all the personality variables at each voxel. The original model is partitioned so that $\boldsymbol{\beta} = [\boldsymbol{\beta}_1^T : \boldsymbol{\beta}_2^T]$. For the null hypothesis $H_0 : \beta_1 = 0$ the full partitioned model is

$$\mathbf{Y} = [X_1 \vdots X_2] \begin{bmatrix} \boldsymbol{\beta}_1 \\ \dots \\ \boldsymbol{\beta}_2 \end{bmatrix} + \boldsymbol{\epsilon}, \tag{6.21}$$

also $p = \text{rank}(X)$ and $p_2 = \text{rank}(X_2)$. When H_0 is true,

$$\mathbf{Y} = X_2\boldsymbol{\beta}_2 + \boldsymbol{\epsilon}. \tag{6.22}$$

The two models have residual sums of squares $S(\boldsymbol{\beta})$ and $S(\boldsymbol{\beta}_2)$ leading to an F-test defined as

$$F = \frac{(S(\boldsymbol{\beta}_2) - S(\boldsymbol{\beta}))/(p - p_2)}{S(\boldsymbol{\beta})/(J - p)}, \tag{6.23}$$

which can be compared to the F-distribution with numerator $(p - p_2)$ and denominator $(J - p)$ degrees of freedom.

To test for significant differences between the quartile levels of the personality variables (conditions 1 to 4), a t-test is used. For a test between conditions one and two, the contrast is defined as $\mathbf{c} = [-1\ 1\ 0\ 0\ \dots]$ (i.e. condition two is activated compared to condition one), giving a t-test for the jth voxel as

$$t_j = \frac{\mathbf{c}.\boldsymbol{\beta}_j}{\epsilon_j} \tag{6.24}$$

where $\epsilon_j = \sqrt{\widehat{\sigma}^2\mathbf{c}^T(X^TX)^{-1}\mathbf{c}}$ and the test has $J - p$ degrees of freedom. $\widehat{\sigma}^2$ is the residual sum of squares divided by the $J - p$ degrees of freedom.

Looking at the significance of these tests in every voxel one has to take into account the highly dependent nature of the image data. The next section presents the theory on how this dependency problem is accounted for within the SPM99 framework.

6.4.2 Voxel and Cluster Levels

To look for areas of activation (or deactivation), each voxel was tested individually, for significance, across the twenty subjects. Due to the highly intercorrelated dependent nature of the data, a corrective procedure (Worsley, 1994, 1995; Friston et al., 1995b, 1994), based on Gaussian random field theory, was implemented, within SPM99. The theory is summarised in Frackowiak et al. (1997).

Voxel-level analysis allows for the detection of individual significant voxels. The maximum Z score is compared to a threshold (t). The probability of Z_{max} being greater than t is calculated using Euler characteristics giving for high t (Worsley et al., 1995)

$$Pr(Z_{max} \geq t) \approx E[\chi_t], \tag{6.25}$$

where

$$E[\chi_t] = \lambda(V)|\Lambda|^{\frac{1}{2}}(2\pi)^{-\frac{D+1}{2}} He_D(t) e^{-\frac{t^2}{2}}.$$

$\lambda(V)$ is the volume being analysed, $He_D(t)$ is a Hermite polynomial of degree D and Λ is the variance-covariance matrix of partial derivatives in the D directions.

Inference at the cluster-level, used for this study, identifies significant activations in groups of voxels (called clusters). Rather than thresholding for a significant voxel, the spatial extent (S) of a group of activated voxels was considered, to identify activation clusters. The number of voxels in the cluster was compared to the expected number of voxels per cluster. This reduces the localising power of the test from that of a voxel, to a region of voxels, but conversely increases the power (Friston et al., 1994).

Both the spatial extent and the peak intensity (H) are compared to some threshold (s_0 and h_0 respectively). With a null hypothesis, of pure noise, the probability of rejecting a cluster is

$$Pr(\text{rejection}) = 1 - e^{-E[M_t(V)]} Pr_{joint}, \tag{6.26}$$

where $Pr_{joint} = Pr(n_u \geq s_0) + Pr(H \geq h_0) - Pr(n_u \geq S_0, H \geq h_0)$, for an area n_u with u intensity thresholds. Equations 6.25 and 6.26 are used to calculate probabilities for the significance of a voxel, or cluster, whilst taking into account the dependent nature of the data.

6.4.3 Contrasts

A two-sided t-test was used to investigate contrasts between the various quartile levels within each model. Every pairwise comparison of Q1, Q2, Q3 and Q4 was tested with only the significant results presented. For example Q1 was contrasted with Q2 as both an activation and deactivation (hence the two-tailed test). A significance level of $\alpha = 0.05$ was

Coordinates	Cluster Size	Region	Percentage in that region
52 -40 12	131	Temporal_Sup_R	55.73
		Temporal_Mid_R	23.66
		SupraMarginal_R	20.61

Table 6.1: Example output from the automated anatomical labelling with SPM interface

used to identify significant clusters. Due to multiple comparison testing between the four quartile levels, a Bonferroni correction (Cliff, 1987) was implemented. The Bonferroni correction is generally considered a conservative approach. All significant results are presented, with clusters significant at the Bonferroni level highlighted.

6.4.4 Locations of Activations

As this study uses functional images rather than structural images, atlases were used to anatomically locate the clusters. This was achieved by implementing three different approaches, the Talairach Space Utility (www.ihb.spb.ru/~pet_lab/TSU/TSUMain.html), automated anatomical labelling in SPM (Tzourio-Mazoyer et al., 2002) and MRIcro (www.cla.sc.edu/psyc/faculty/rorden/mricro.html).

The first most subjective approach was the Talairach Space Utility, which is based on the Talairach and Tournoux (1988) atlas. The TSU plotted the cluster onto the Talariach Atlas from which the anatomic locations could be read off. For example a cluster of deactivation associated with reward dependence, with 131 voxels, gives the output shown in Figure 6.3. This is quite difficult to interpret, even with the Talairach and Tournoux (1988) atlas on hand.

The second approach using the automated SPM interface is based on the atlas developed by Tzourio-Mazoyer et al. (2002). The authors anatomically labelled the MNI single subject brain then implemented a system of calculating the percentage of clusters in each of the anatomical regions. For example for the same cluster of deactivation associated with reward dependence, with 131 voxels, the output from the automated SPM interface is presented in Table 6.1. Unlike the other methods this method gives the percentage of the cluster that is in the particular region.

The third approach is also based on the Tzourio-Mazoyer et al. (2002) atlas but implemented within the MRIcro program. The MRIcro program has used the anatomical labelling developed by Tzourio-Mazoyer et al. (2002) to create a map, as shown in Figure 6.4, in which the clusters can be compared to obtain anatomic locations.

There are anatomical differences between individual brains both in size and shape. This was the reason for using the stereotactic normalisation to align brains into a standard

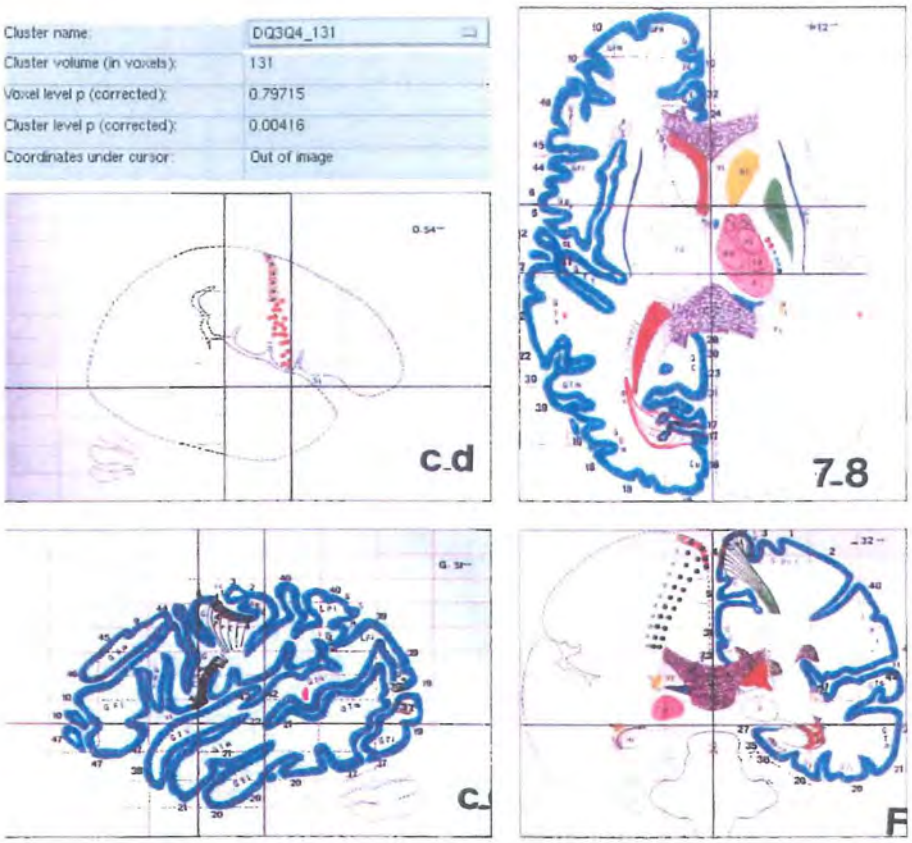


Figure 6.3: Talairach space utility output with cluster location in red.

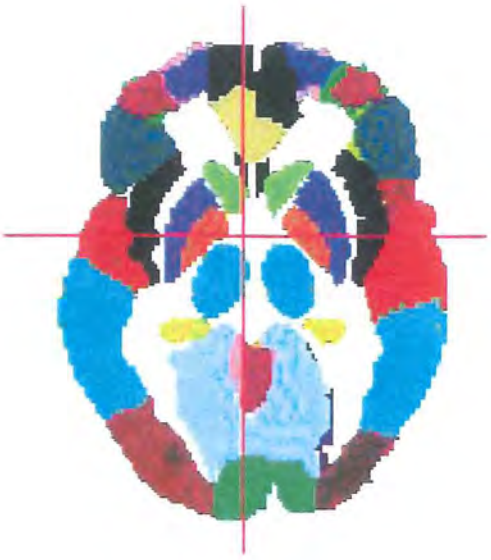


Figure 6.4: One slice of the AAL map in the program MRIcro.

space. This means that when analysing the voxels, the same voxel across all brains should correspond to the same anatomic location in all the brains. The atlases used to calculate the anatomic locations of the resultant clusters are based on a single brain. This single brain, whilst in the same space as the analysed brains, will contain more individuality than say an average normal brain. So calculating the locations based on one brain is not ideal and has limitations. The ideal solution would be to use a standard anatomical brain map that was based on an average of a number of different brains.

The MNI brain space that the images are stereotactically normalised to is based on the average of 305 brains; however there is not a corresponding anatomical map for this brain space that could be used to work out the locations of the results. In most studies in brain imaging fMRI is the imaging modality of choice and in the process of taking the fMRI image generally a standard MRI image is obtained for use in locating the activations. With SPECT images there are no such corresponding structural brain images so the single brain atlases are the only option for locating the activations. It is hoped in future work that atlases based on the MNI brain space will be readily available for this use.

Whilst an atlas based on a so-called average brain, would have been optimal, only atlases based on single subjects were available for this study. Given that SPM and MRIcro approaches are based on the same atlas, the Talairach and Tournoux (1988) atlas was used as an additional check for cluster location. Only two of the forty-two clusters found, disagreed in terms of location as specified by the three methods.

6.5 Results

The sample contained 18 right-handers and 2 left-handers. Initial analyses, using SPM two sample t-tests, were unable to detect any significant left/right handedness effect.

The sample distributions of the TCI personality traits are presented as side-by-side boxplots in Figure 6.5. From Figure 6.5 it can be seen that all the personality traits are non-gaussian and skewed, indeed all the traits except harm avoidance and reward dependence are right skewed. The range of the medians across all the temperament traits is within $0.40 - 0.60$, indicating a central tendency for these traits. Table 6.2 reports the lower quartile, median, upper quartile and direction of skew for each personality trait. In the presence of skew in the personality variables, the use of quartiles provides more power to detect associations between personality and blood flow. This type of quartile grouping also allowed for possible non-linear relationships to be investigated.

Significant clusters of activation (relationship between increasing level of trait and increasing blood flow) or deactivation (relationship between decreasing level of trait and increasing level of blood flow) were found in relation to all seven TCI traits.

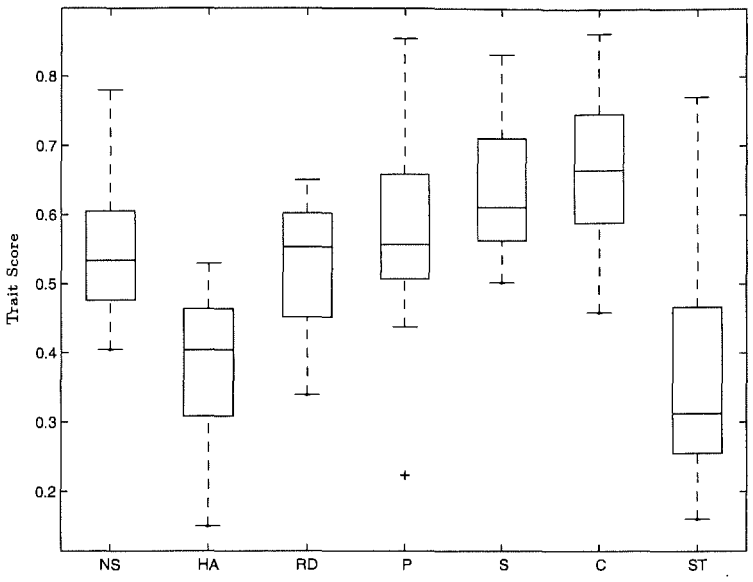


Figure 6.5: Distribution of personality scores for the normal males.

	NS	HA	RD	P	S	C	ST
Lower Quartile	0.476	0.308	0.452	0.507	0.563	0.589	0.255
Median	0.534	0.404	0.553	0.557	0.611	0.665	0.313
Upper Quartile	0.606	0.464	0.602	0.658	0.711	0.746	0.467
Nature of Skew	Right	Left	Left	Right	Right	Right	Right

Table 6.2: Distribution of personality traits.

The results for the seven models are presented in Tables 6.3-6.9. These tables show the significant clusters found for each TCI trait, grouped by the different quartile contrasts. The expected voxels per cluster (N_E) for the model are shown below the trait name. These were calculated within SPM99 using Euler characteristics (Frackowiak et al., 1997). The first column gives the contrast (i.e. Q2 Q4 is a contrast between the Q2 and Q4 quartile groups), whether it is an activation or deactivation and below this, the location of the cluster in anatomical terms. The general region is given, along with the specific parts of the region that are in the cluster. The second column shows firstly, the coordinates of the strongest voxel (by t-value) in the cluster in MNI space (x,y,z), the number of voxels in the cluster (N) and finally the p-value associated with observing a cluster of size N compared to N_E. The p-values in bold are those that are below the Bonferroni corrected significance level ($\alpha \approx 0.008$). Twenty-eight of the 42 clusters (66.7%) found are significant at this conservative level.

Within each trait, clusters were checked for overlap, then the average rCBF for the

cluster was plotted versus the quartile level or group. The contrasts reveal the significant differences that cannot be obtained by an overall linear test. The graphs demonstrate the non-linear relationship seen between blood flow and personality groupings. The locations of the clusters are plotted on a smoothed average brain with the activation and cluster sizes shown below each figure.

6.5.1 Results for Novelty Seeking

Novelty seeking appears to be significantly related to blood flow, as shown in Table 6.3. Within the novelty seeking model, a contrast between Q2 and Q4 showed a significant relationship with blood flow in the left precentral and post central gyrus ($N = 337$). This implies that as the levels of novelty seeking increase from Q2 to Q4, blood flow is significantly increased in the left central region. The average rCBF in this cluster is plotted versus quartile group in Figure 6.6(a), showing a general increase in blood flow from Q2 to Q4. Whilst Q1 is not significantly higher in rCBF than Q2 there does appear to be a decrease.

Five significant clusters of deactivation were found. A cluster of size $N = 611$, found in the left precuneus, calcarine and lingual gyrus, has a significant deactivation from the Q1 to Q3 quartile groups. Graphing average rCBF in the cluster versus quartile group (Figure 6.6(b)) showed a general decrease in average rCBF from Q1 to Q3 with no significant difference between Q3 and Q4, though there appears to be an increase.

Clusters $N = 185$ and $N = 97$ had the same general trend in rCBF demonstrated in Figures 6.6(c) and 6.6(d). The larger cluster was located in the right temporal lobe (inferior temporal gyrus), right occipital lobe (fusiform gyrus) and the right limbic lobe (middle and superior temporal pole). The smaller cluster was located in the right middle and superior temporal gyrus and the right superior temporal pole gyrus. Both these clusters exhibit no significant change from Q1 to Q2 and then a general decrease in rCBF from Q2 to Q4. The average rCBF for Q4 is significantly smaller than that for Q2.

Clusters $N = 1010$ and $N = 180$ overlap with 170 voxels in common. Cluster $N = 1010$ had a significant decrease in rCBF from Q1 to Q4 and was located in the left temporal and limbic lobes. Specifically the regions of the inferior and middle temporal gyrus and the middle temporal pole gyrus were involved. Cluster $N = 180$, which is almost entirely contained in the larger cluster, shows a significant decrease in average rCBF from Q2 to Q4 and spanned the left temporal lobe (inferior and middle gyrus) and limbic lobe (middle temporal pole). This indicates that in the overlapping region (94% of $N = 180$) the average rCBF for quartile group Q4 is significantly lower than that of both Q1 and Q2. This average rCBF is shown graphically in Figures 6.6(e) and 6.6(f), the first shows the average rCBF in cluster $N = 1010$ and the second graph shows the average rCBF in

the overlapping region.

Figures 6.16(a) and 6.16(b) show the locations of the clusters on a smoothed average brain (SPM99 template). The second figure clearly shows the overlap between clusters $N = 1010$ and $N = 180$.

Novelty Seeking *(Expected voxels per cluster N_E = 5.920)*	Coordinates	Cluster N	p-value
Activation Q2 Q4			
Left Central Region:	(-40, -20, 68)	337	**0.000**
Precentral gyrus, Postcentral gyrus			
Deactivation Q1 Q4			
Left Temporal Lobe:	(-44, 8, -42)	1010	**0.000**
Inferior, middle temporal gyrus			
Left Limbic Lobe:			
Temporal pole: middle temporal gyrus			
Deactivation Q1 Q3			
Left Parietal Lobe:	(-18, -52, 4)	611	**0.000**
Precuneus			
Left Occipital Lobe:			
Calcarine, lingual gyrus			
Deactivation Q2 Q4			
Right Temporal Lobe:	(32, 30, -36)	185	**0.001**
Inferior temporal gyrus			
Right Occipital Lobe:			
Fusiform gyrus			
Right Limbic Lobe:			
Temporal pole: middle, superior temporal gyrus			
Left Temporal Lobe:	(-46, 2, -48)	180	**0.001**
Inferior, middle temporal gyrus			
Left Limbic Lobe:			
Temporal pole: middle temporal gyrus			
Right Temporal Lobe:	(70, -4, -10)	97	0.037
Middle, superior temporal gyrus			

Table 6.3: Contrast results for novelty seeking.

Novelty Seeking	Cluster		
(Expected voxels per cluster N_E = 5.920)	Coordinates	N	p-value
Right Limbic Lobe:			
Temporal pole: superior temporal gyrus			

Table 6.3: Contrast results for novelty seeking.

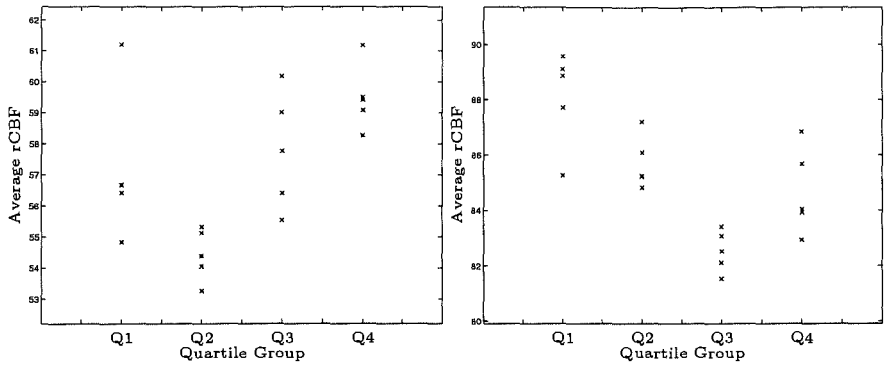
6.5.2 Results for Harm Avoidance

Modelling harm avoidance as a predictor of blood flow identified two clusters with a significant relationship between rCBF and quartile group (Table 6.4). The cluster locations on the smoothed average brain are presented in Figure 6.16(c). The first cluster $N = 191$ showed a significant increase in rCBF between quartile group 1 and quartile group 3. The cluster was located in the right occipital and limbic lobes. The regions specifically involved were the fusiform gyrus, middle and superior temporal pole, and the parahippocampal gyrus. Figure 6.7(a) shows the graph of rCBF versus quartile group. There is clearly an increasing relationship between rCBF and harm avoidance as the quartile groups increased from Q1 to Q3. Q3 is not significantly different from Q4 though the graph showed a general decrease.

The second cluster $N = 390$ has a significant increase in blood flow as harm avoidance increases from Q2 to Q3. This cluster is located in the occipital lobe, involving the middle and superior occipital gyrus. There is a downward trend from Q1 to Q2 and likewise from Q3 to Q4 (see Figure 6.7(b)) but statistically there is no significant difference.

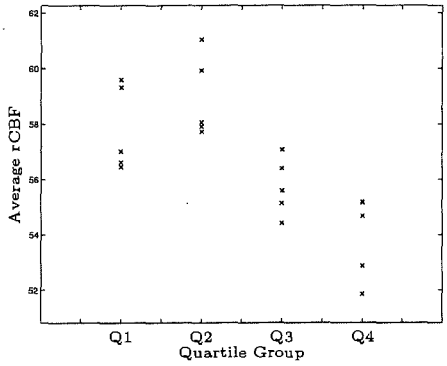
Harm Avoidance	Cluster		
(Expected voxels per cluster N_E = 6.052)	Coordinates	N	p-value
Activation Q1 Q3			
Right Occipital Lobe:	(24, 14, -40)	191	**0.001**
Fusiform gyrus			
Right Limbic Lobe:			
Temporal Pole: middle, superior temporal gyrus			
Parahippocampal gyrus			
Activation Q2 Q3			
Right Occipital Lobe:	(30, -68, 16)	390	**0.000**
Middle, superior occipital gyrus			
Calcarine, cuneus			

Table 6.4: Contrast results for harm avoidance.

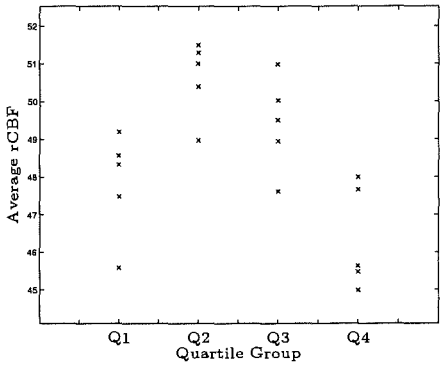


(a) Cluster N=337

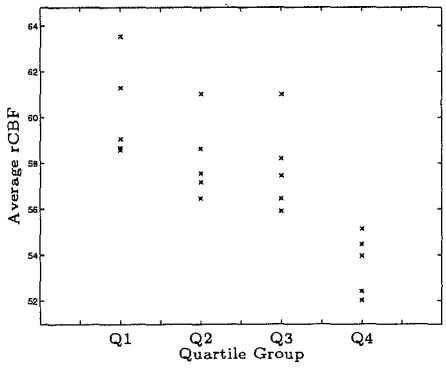
(b) Cluster N=611



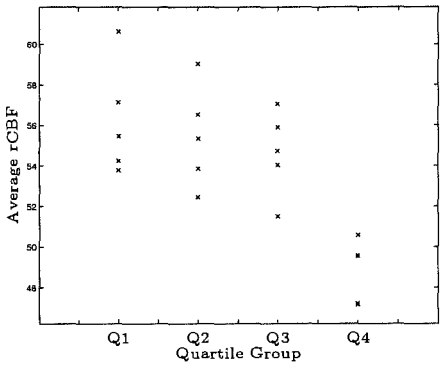
(c) Cluster N=185



(d) Cluster N=97

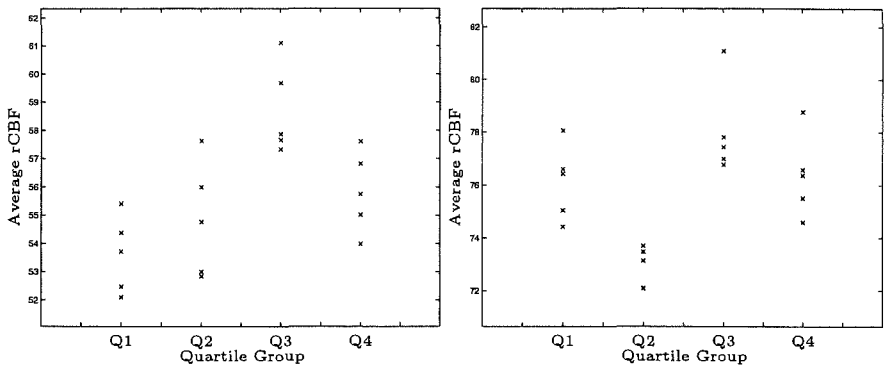


(e) Cluster N=1010



(f) Intersection of Cluster N=1010 and N=180

Figure 6.6: Average rCBF versus quartile group for novelty seeking.



(a) Cluster N=191 (b) Cluster N=390

Figure 6.7: Average rCBF versus quartile group for harm avoidance.

6.5.3 Results for Reward Dependence

Using the reward dependence model, four clusters were found that had significant relationships between rCBF and the quartile levels of reward dependence. The results are presented in Table 6.5. The activations are shown in Figure 6.16(d) and the deactivations in 6.16(e). These four clusters do not overlap. The first cluster $N = 118$ has a significant activation from Q1 to Q2. It is located in the left frontal lobe (middle frontal gyrus, inferior and middle orbital frontal gyrus). Figure 6.8(a) shows the graph of average rCBF and quartile levels for this cluster. The activation can be clearly seen with Q2, Q3 and Q4 showing no significant differences. This suggests an increase in reward dependence at low levels relates to an increase in rCBF, after this increasing reward dependence had little effect on rCBF.

Reward Dependence	Cluster		
(Expected voxels per cluster N_E = 6.193)	Coordinates	N	p-value
Activation Q1 Q2			
Left Frontal Lobe:	(-42, 54, 0)	118	0.015
Middle frontal gyrus			
Inferior, middle frontal gyrus: orbital part			
Activation Q1 Q3			
Right Temporal Lobe:	(66, -38, -22)	163	**0.002**
Inferior, middle temporal gyrus			
Deactivation Q2 Q4			
Right Occipital Lobe:	(26, -70, 30)	227	**0.000**

Table 6.5: Contrast results for reward dependence.

Reward Dependence	Cluster		
(Expected voxels per cluster N_E = 6.193)	Coordinates	N	p-value
Middle, superior occipital gyrus			
Calcarine, cuneus			
Deactivation Q3 Q4			
Right Temporal Lobe:	(52, -40, 12)	131	0.008
Middle, superior temporal gyrus			
Right Parietal Lobe:			
Supramarginal gyrus			

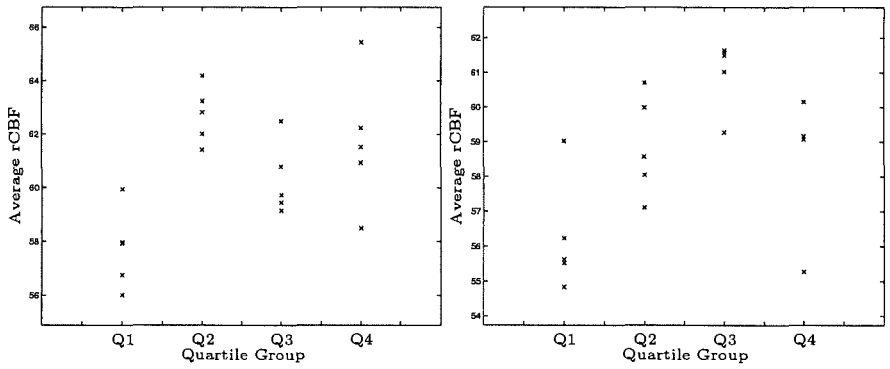
Table 6.5: Contrast results for reward dependence.

The second cluster $N = 163$ located in the right temporal lobe (inferior and middle temporal gyrus) showed significantly increased rCBF from Q1 to Q3. Figure 6.8(b) shows that there is a general increase in rCBF with increasing reward dependence from Q1 through to Q3, with no significant difference between Q3 and Q4.

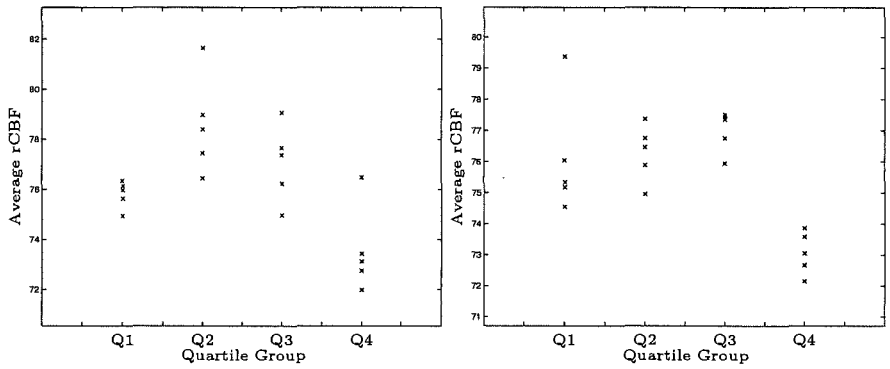
Deactivations were found in the right temporal (middle and superior temporal gyrus) and occipital (middle and superior occipital gyrus, calcarine and cuneus) lobes along with the supramarginal gyrus (see Figure 6.16(e)). These deactivations were associated with a contrast of Q4 to both Q2 and Q3. Figure 6.8(c) shows that, in the first cluster $N = 227$, Q4 is significantly lower than Q2. In the second cluster $N = 131$, Q4 is significantly lower than Q3 (see Figure 6.8(d)).

6.5.4 Results for Persistence

Increasing levels of persistence, like reward dependence, were related to activations in the temporal lobe (left inferior temporal gyrus), however there were also activations in the parietal, occipital and limbic lobes, and deactivations in the parietal, temporal and frontal lobes, as well as the rolandic operculum, heschl gyrus and insula. This is illustrated in Figure 6.16(f) with the clusters presented in Table 6.6. The five clusters found were all distinct with no overlap. The graphs (see Figure 6.9) show the non-linear relationships. Two clusters ($N = 797$ and $N = 136$) show a general upward trend in rCBF as levels of persistence increase from Q1 to Q3. A drop in blood flow can be seen from Q3 to Q4 but this is not significant. The last 3 clusters ($N = 173$, $N = 128$, $N = 123$) show little or no change in rCBF from Q1 to Q2 then a significant drop from Q2 to Q3, with no significant difference between Q3 and Q4.



(a) Cluster N=118 (b) Cluster N=163



(c) Cluster N=227 (d) Cluster N=131

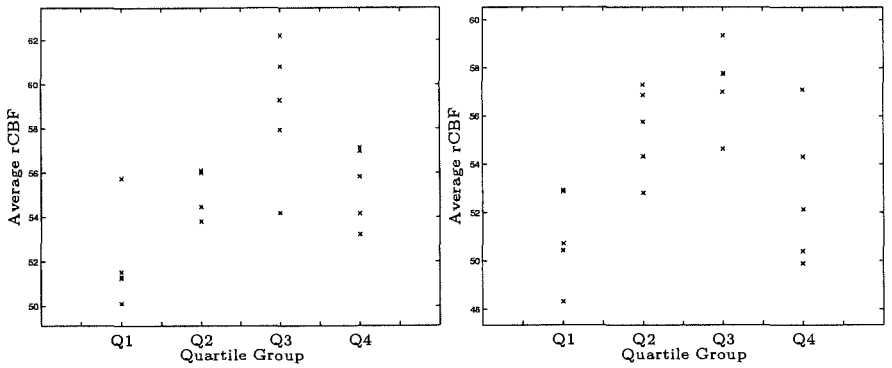
Figure 6.8: Average rCBF versus quartile group for reward dependence.

Persistence	Cluster		
(Expected voxels per cluster N_E = 6.035)	Coordinates	N	p-value
Activation Q1 Q3			
Left Temporal Lobe:	(-44, -8, -46)	797	**0.000**
Inferior temporal gyrus			
Left Parietal Lobe:			
Temporal pole: middle temporal gyrus			
Left Occipital Lobe:			
Fusiform gyrus			
Right Occipital Lobe:	(24, -10, -46)	136	**0.006**
Fusiform gyrus			
Right Limbic Lobe:			
Parahippocampal gyrus			
Deactivation Q2 Q3			
Left Temporal Lobe:	(-48, -40, 26)	173	**0.001**
Superior temporal gyrus			
Left Parietal Lobe:			
Inferior parietal lobule, supramarginal gyrus			
Right Frontal Lobe:	(60, 28, 14)	128	0.008
Inferior frontal gyrus, triangular part			
Inferior frontal gyrus, opercular part			
Right Central Region:	(38, -14, 18)	123	0.011
Rolandic Operculum			
Right Temporal Lobe:			
Heschl gyrus			
Right Other:			
Insula			

Table 6.6: Contrast results for persistence.

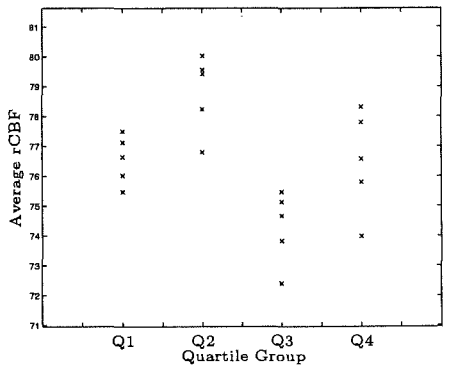
6.5.5 Results for Self Directedness

Table 6.7 shows the results for self directedness as a predictor of blood flow. Higher levels of self directedness were associated with one cluster of activation in the left frontal lobe

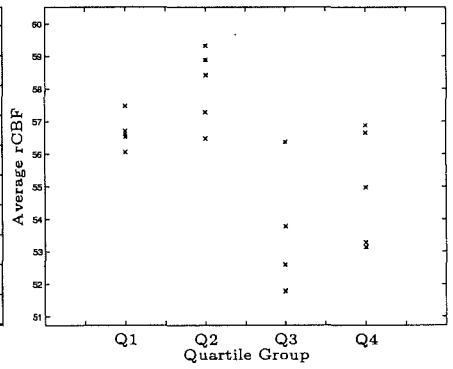


(a) Cluster N=797

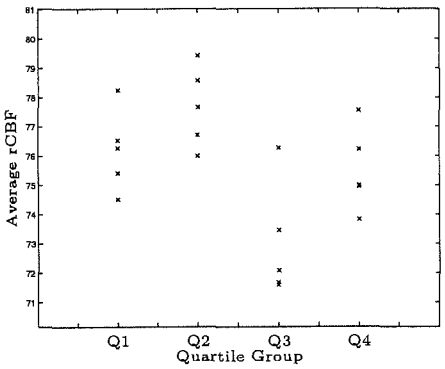
(b) Cluster N=136



(c) Cluster N=173



(d) Cluster N=128



(e) Cluster N=123

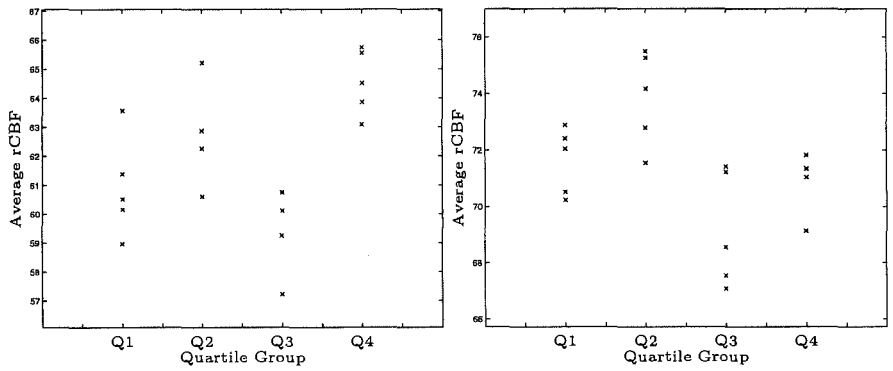
Figure 6.9: Average rCBF versus quartile group for persistence.

(middle frontal gyrus, inferior and middle orbital frontal gyrus, inferior triangular frontal gyrus). Figure 6.10(a) shows that there is a significant increase in blood flow from the third quartile group to the fourth. There appears to be a drop in blood flow from Q2 to Q3 but this is not statistically significant.

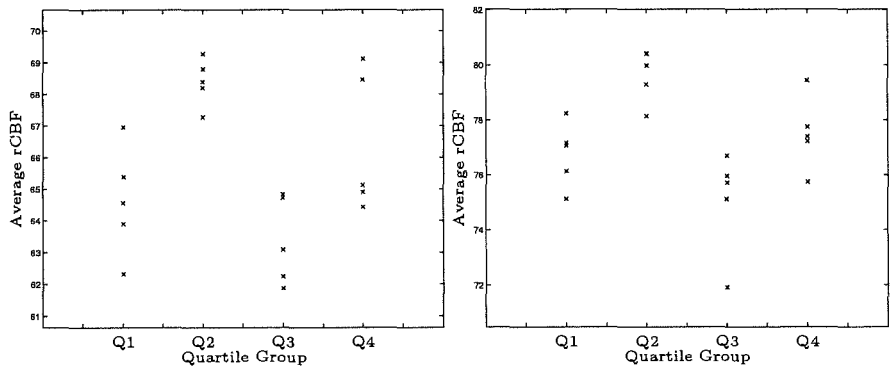
Three deactivation clusters were also related to higher levels of self directedness. These clusters exhibit a similar pattern in rCBF (see Figure 6.10). No significant difference is found between Q1 and Q2, likewise between Q3 and Q4. The Q2 group had significantly more blood flow than Q3. The largest cluster ($N = 276$) was located in the right precentral gyrus and right middle frontal gyrus. The second largest, with 193 voxels, was located in the right inferior temporal and fusiform gyrus. The third cluster ($N = 178$) was located in the left middle and superior temporal gyrus. The cluster locations are shown in Figure 6.17(a) with activation in red and deactivation in green.

Self Directedness		Cluster	
(Expected voxels per cluster $N_E = 6.331$)	Coordinates	N	p-value
Activation Q3 Q4			
Left Frontal Lobe:	(-48, 44, -2)	100	0.041
Inferior frontal gyrus, triangular part			
Inferior frontal gyrus, orbital part			
Middle frontal gyrus			
Middle frontal gyrus, orbital part			
Deactivation Q2 Q3			
Right Central Region:	(52, -2, 42)	276	**0.000**
Precentral gyrus			
Right Frontal Lobe:			
Middle frontal gyrus			
Right Temporal Lobe:	(62, -30, -20)	193	**0.001**
Inferior temporal gyrus			
Right Occipital Lobe:			
Fusiform gyrus			
Left Temporal Lobe:	(-46, -40, 12)	178	**0.001**
Middle, superior temporal gyrus			

Table 6.7: Contrast results for self directedness.



(a) Cluster N=100 (b) Cluster N=276



(c) Cluster N=193 (d) Cluster N=178

Figure 6.10: Average rCBF versus quartile group for self directedness.

6.5.6 Results for Cooperativeness

Cooperativeness exhibited the largest cluster (N= 3108) across all the personality traits and also had the most clusters detected. Cooperativeness also had two clusters upon which the three location methods did not agree. These clusters are both deactivations, the first being a contrast between Q2 and Q3 ($N = 258$) and the second a contrast between Q2 and Q4 ($N = 128$).

The largest cluster ($N = 3108$) was related to a contrast between Q1 and Q2. It was located in the frontal lobes, both left and right. This cluster intersects with three other clusters. Clusters $N = 470$ and $N = 115$ overlap with $N = 3108$ and have 102 voxels that are common to all three. $N = 115$ is entirely contained in $N = 3108$ and $N = 437$ has 335 voxels contained in $N = 3108$. The two smaller clusters are located in the orbital part of the left inferior frontal gyrus and the temporal pole of the left superior temporal gyrus. Cluster $N = 115$ shows a significant activation from Q1 to Q4, $N = 437$ has a significant activation from Q1 to Q3. This means that in the overlap of all three regions rCBF in group Q1 is significantly lower than groups Q2, Q3 and Q4 (see Figure 6.11(a)). The graph of rCBF in cluster $N = 3108$ is shown in Figure 6.11(b), cluster $N = 470$ in Figure 6.11(c) and cluster $N = 115$ in Figure 6.11(d).

Cluster $N = 3108$ also overlaps with cluster $N = 103$, which is entirely contained in the larger cluster. In this overlapping region there is a significant activation from Q1 to Q2 then a significant deactivation from Q2 to Q4. This is shown in Figure 6.11(e). The cluster is located in the gyrus rectus in the left frontal lobe.

A small cluster of voxels found in the right frontal lobe shows a significant activation between Q1 and Q2. This cluster $N = 93$ is located in the triangular part of the right inferior frontal gyrus and the right middle frontal gyrus. Average rCBF for the cluster is plotted versus quartile group in Figure 6.11(f), clearly showing the significant activation from Q1 to Q2. There appears to be a general decreasing trend after this but not statistically so.

A third cluster showing significant activations from Q1 to Q2 was found in the right sub cortical gray nuclei with size $N = 92$. This cluster overlaps with a cluster of deactivation from Q2 to Q3 ($N = 333$). The overlapping region is 33 voxels large. In this region there is both a significant activation from Q1 to Q2 and a significant deactivation from Q2 to Q3. So at low levels of cooperativeness there is an increased blood flow with increased cooperativeness, however after Q2 this reverses and increasing cooperativeness is related to decreased blood flow. There is no significant difference from Q3 to Q4. The relationship between average rCBF and cooperativeness for cluster $N = 92$ is shown in Figure 6.12(a), for cluster $N = 333$ in Figure 6.12(b) and for the overlapping region in Figure 6.12(c).

The last activation found was from Q2 to Q3. This cluster was of size $N = 253$ and

was located in the left inferior, middle and middle temporal pole gyrus. Figure 6.12(d) shows that as cooperativeness increases from Q1 through to Q4, there is no significant change until the significant increase from Q2 to Q3 and then no significant change from Q3 to Q4.

Three overlapping clusters of deactivation were found in the left parietal lobe, with 74 voxels common to all three. The first cluster ($N = 139$) is located in the left inferior and superior parietal lobes. The second cluster ($N = 478$) is located in the left inferior and superior parietal lobes and the left postcentral gyrus. The third cluster ($N = 503$) spreads through the left and right superior parietal lobe and precuneus, the left inferior parietal lobe and the right cuneus. Cluster $N = 139$ was significantly deactivated from Q1 to Q3 (see Figure 6.13(a)), cluster $N = 478$ exhibited a significant deactivation from Q2 to Q3 (Figure 6.13(b)) and cluster $N = 503$ showed a significant deactivation from Q2 to Q4 (Figure 6.13(c)). In the overlapping region of all three clusters Q3 and Q4 are both significantly lower in rCBF than Q1 and Q2 (see Figure 6.13(d)).

Cluster $N = 503$ also overlaps with a small cluster of deactivation ($N = 93$) in the left superior parietal gyrus, precuneus and right precuneus. The overlapping region has 56 voxels. The small cluster exhibits a deactivation from Q3 to Q4 and is shown in Figure 6.14(c). The intersecting region is shown in Figure 6.14(d) where both Q2 and Q3 have significantly higher blood flow than Q4.

Two overlapping clusters exhibiting deactivations were found in the left central region and left frontal region. The first cluster ($N = 112$) shows a deactivation from Q1 to Q3 in the left precentral gyrus and left middle frontal gyrus, shown in Figure 6.13(e). The second cluster $N = 258$ is also located in the left precentral gyrus and left middle frontal gyrus but also spreads through the left subcortical gray nuclei (caudate nucleus) and the occipito fasiculus, shown in Figure 6.13(f)). The overlapping region contains 37 voxels (see Figure 6.14(a)).

Cluster $N = 258$ from above, and cluster $N = 128$, a cluster of deactivated voxels comparing Q2 to Q4, share 12 voxels in common located in the left temporal lobe, subcortical gray matter, insula and pyramidal pathways. The graph of average rCBF versus quartile groups for cluster $N = 128$ is shown in Figure 6.14(b).

A cluster of $N = 153$ voxels was found in the right postcentral gyrus, superior parietal lobe and precuneus showing a significant deactivation from Q2 to Q3.

A large cluster ($N = 584$) was found that showed a significant deactivation from Q2 to Q4. In this region average rCBF appears to rise from Q1 to Q2 (not significantly) and then fall from Q2 through to Q4 with rCBF significantly lower in Q4 than Q2 (see Figure 6.14(e)). This cluster is spread through the left and right occipital and limbic lobes, left and right cerebellum, left parietal lobe, left sub cortical gray nuclei and vermis.

The last cluster associated with a significant decrease in blood flow from Q2 to Q4 is

located in the right temporal and occipital lobes. It has a size of 100 voxels and exhibits a similar pattern of average rCBF to that in cluster $N = 584$ above (see Figure 6.14(f)).

Figure 6.18(a) shows the activations associated with a contrast between Q1 and Q2 in red, Q1 and Q3 in green, and Q1 and Q4 in blue, which clearly shows the overlapping region for clusters $N = 3108$, $N = 470$, and $N = 115$. The overlap between $N = 3108$ (AQ1Q2) and $N = 103$ (DQ2Q4) can be seen in Figure 6.18(b) which shows the locations of clusters $N = 3108$ and all the clusters associated with DQ2Q4. Figure 6.18(c) shows the two larger clusters associated with DQ2Q3 (namely $N = 478$ and $N = 333$) and all the clusters associated with AQ1Q2. This clearly shows the overlap between clusters $N = 333$ (DQ2Q3) and $N = 92$ (AQ1Q2). The location of cluster $N = 253$ (AQ2Q3) is shown in Figure 6.18(d). Figure 6.18(e) shows the locations of the all the clusters associated with DQ1Q3, DQ2Q3 and DQ2Q4. The overlap between clusters $N = 139$ (DQ1Q3), $N = 478$ (DQ2Q3) and $N = 503$ (DQ2Q4) can be clearly seen. Clusters $N = 584$, $N = 503$ (DQ2Q4) and $N = 93$ (DQ3Q4) are shown in Figure 6.18(f), with overlap between clusters $N = 503$ and $N = 93$ demonstrated.

Cooperativeness		Cluster	
(Expected voxels per cluster $N_E = 5.398$)	Coordinates	N	p-value
Activation Q1 Q2			
Left Frontal Lobe:	(-44, 32, -12)	3108	**0.000**
Inferior frontal gyrus, triangular part			
Inferior frontal gyrus, orbital part			
Middle frontal gyrus, orbital part			
Superior frontal gyrus, orbital part			
Gyrus rectus			
Right Frontal Lobe:			
Inferior frontal gyrus, orbital part			
Middle frontal gyrus, orbital part			
Superior frontal gyrus, orbital part			
Gyrus rectus			
Right Frontal Lobe:	(60, 32, 16)	93	0.031
Inferior frontal gyrus, triangular part			
Middle frontal gyrus			
Right Sub Cortical Gray Nuclei:	(26, 10, 12)	92	0.033
Caudate nucleus			
Putamen			

Table 6.8: Contrast results for cooperativeness.

Cooperativeness		Cluster	
(Expected voxels per cluster $N_E = 5.398$)	Coordinates	N	p-value
Activation Q1 Q3			
Left Frontal Lobe:	(-44, 32, -14)	470	**0.000**
Inferior frontal gyrus, orbital part			
Left Limbic Lobe:			
Temporal pole: superior temporal gyrus			
Activation Q1 Q4			
Left Frontal Lobe:	(-44, 34, -18)	115	0.009
Inferior frontal gyrus, orbital part			
Left Limbic Lobe:			
Temporal pole: superior temporal gyrus			
Activation Q2 Q3			
Left Temporal Lobe:	(-70, -10, -18)	253	**0.000**
Inferior, middle temporal gyrus			
Left Limbic Lobe:			
Temporal pole: middle temporal gyrus			
Deactivation Q1 Q3			
Left Parietal Lobe:	(-22, -64, 53)	139	**0.003**
Inferior, superior parietal lobule			
Left Central Region:	(-32, 8, 34)	112	0.011
Precentral gyrus			
Left Frontal Lobe:			
Middle frontal gyrus			
Deactivation Q2 Q3			
Left Central Region:	(-22, -64, 52)	478	**0.000**
Postcentral gyrus			
Left Parietal Lobe:			
Inferior, superior parietal lobule			
Right Sub Cortical Gray Nuclei:	(6, 0, 16)	333	**0.000**

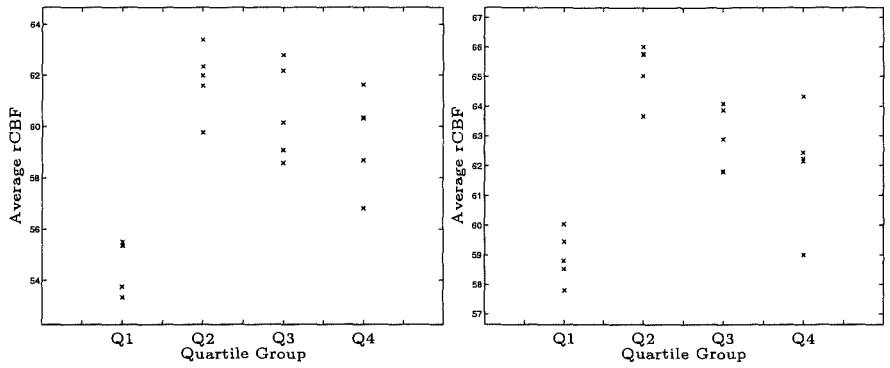
Table 6.8: Contrast results for cooperativeness.

Cooperativeness	Cluster		
(Expected voxels per cluster $N_E = 5.398$)	Coordinates	N	p-value
Caudate nucleus			
Left Central Region:	(-28, 2, 34)	258	**0.000**
Precentral gyrus			
Left Frontal Lobe:			
Middle frontal gyrus			
Left Sub Cortical Gray Nuclei:			
Caudate nucleus			
Left Other:			
Occipito fasciculus			
Right Central Region:	(18, -52, 58)	153	**0.001**
Postcentral gyrus			
Right Parietal Lobe:			
Superior parietal lobule			
Precuneus			
Deactivation Q2 Q4			
Left Parietal Lobe:	(4, -38, 0)	584	**0.000**
Precuneus			
Left Occipital Lobe:			
Lingual gyrus			
Left Limbic Lobe:			
Hippocampus, Posterior cingulum			
Left Sub Cortical Gray Nuclei:			
Thalamus			
Left Other:			
Cerebellum 4 5			
Right Occipital Lobe:			
Lingual gyrus			
Right Limbic Lobe:			
Posterior cingulum			
Right Other:			
Cerebellum 3			
Other:			

Table 6.8: Contrast results for cooperativeness.

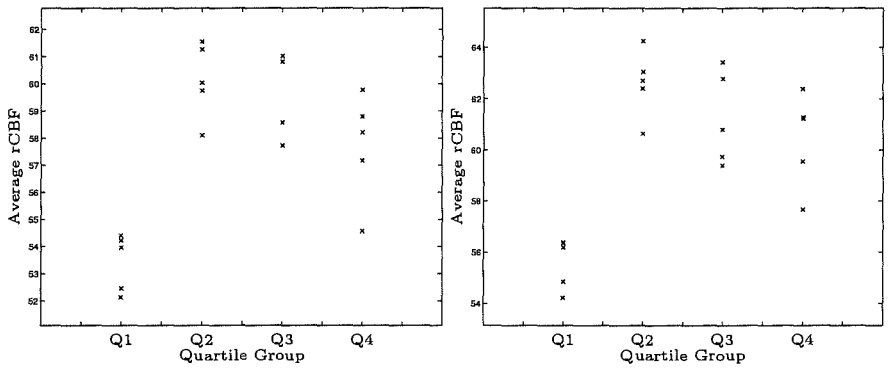
Cooperativeness		Cluster	
(Expected voxels per cluster N_E = 5.398)	Coordinates	N	p-value
Vermis 3, Vermis 4 5			
Left Parietal Lobe:	(-20, -58, 60)	503	**0.000**
Inferior, superior parietal lobule			
Precuneus			
Right Parietal Lobe:			
Superior parietal lobule			
Precuneus			
Right Occipital Lobe:			
Cuneus			
Left Temporal Lobe:	(-24, -26, 14)	128	**0.005**
Heschl gyrus			
Left Sub Cortical Gray Nuclei:			
Thalamus			
Left Other:			
Insula, Pyramidal pathways			
Left Frontal Lobe:	(0, 48, -26)	103	0.017
Gyrus rectus			
Right Temporal Lobe:	(46, -76, -4)	100	0.021
Inferior, middle temporal gyrus			
Right Occipital Lobe:			
Inferior, middle occipital gyrus			
Deactivation Q3 Q4			
Left Parietal Lobe:	(8, -82, 50)	93	0.031
Superior parietal gyrus, Precuneus			
Right Parietal Lobe:			
Precuneus			

Table 6.8: Contrast results for cooperativeness.



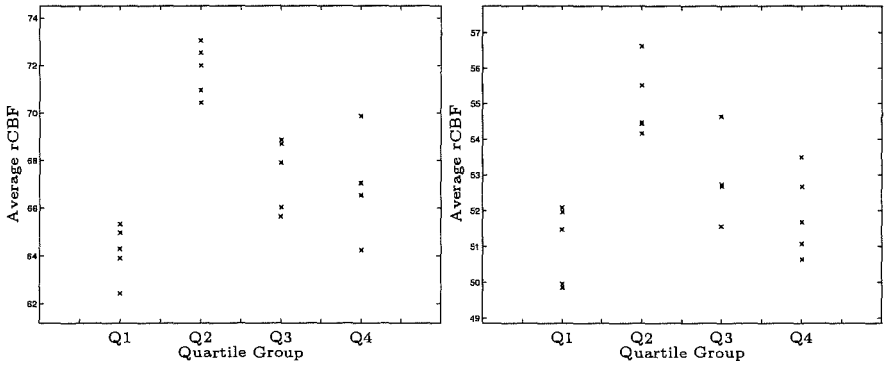
(a) Cluster N=3108, N=470 and N=115

(b) Cluster N=3108



(c) Cluster N=470

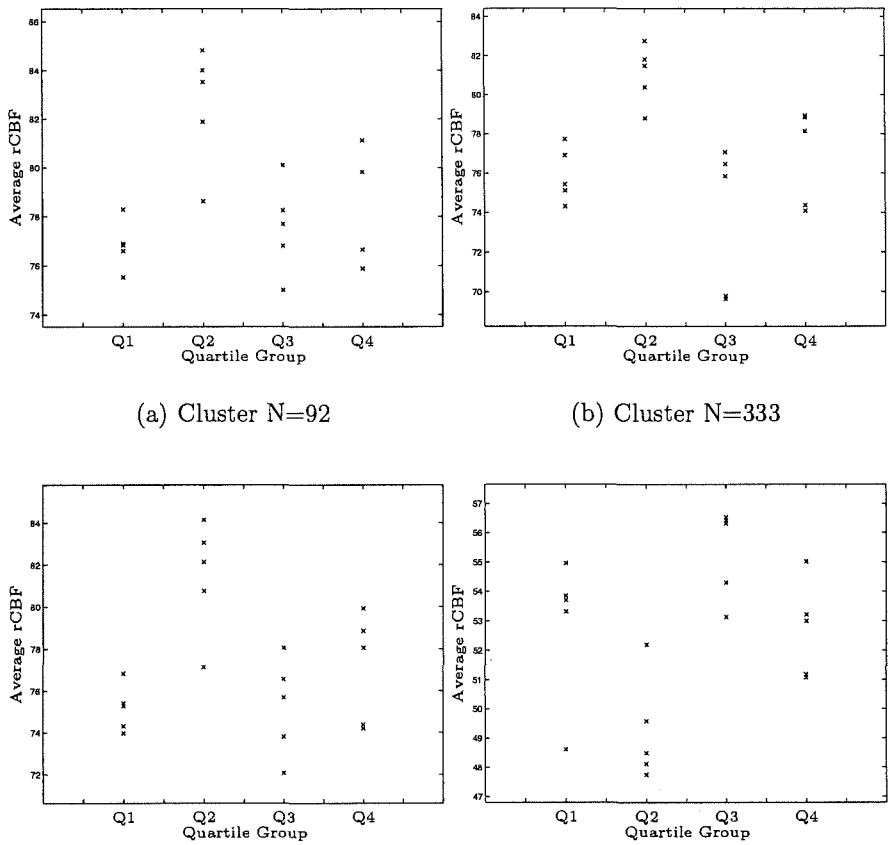
(d) Cluster N=115



(e) Cluster N=3108 and N=103

(f) Cluster N=93(AQ1Q2)

Figure 6.11: Average rCBF versus quartile group for cooperativeness.



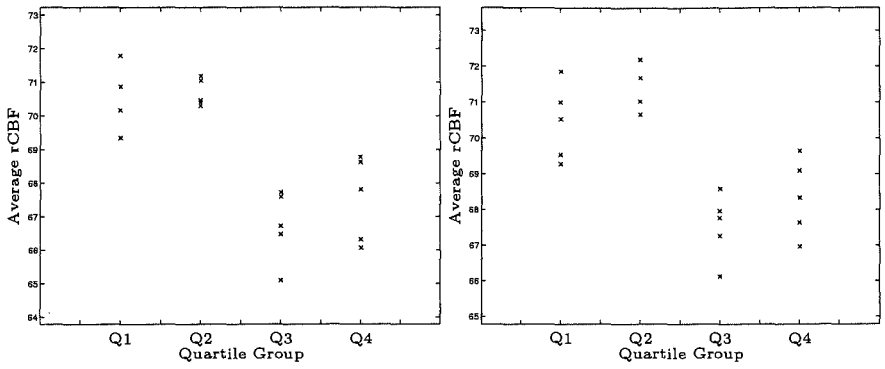
(a) Cluster N=92

(b) Cluster N=333

(c) Cluster N=333 and N=92

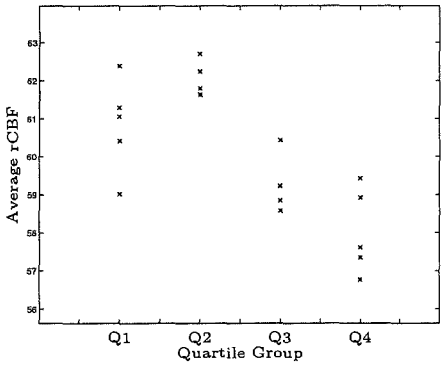
(d) Cluster N=253

Figure 6.12: Average rCBF versus quartile group for cooperativeness.

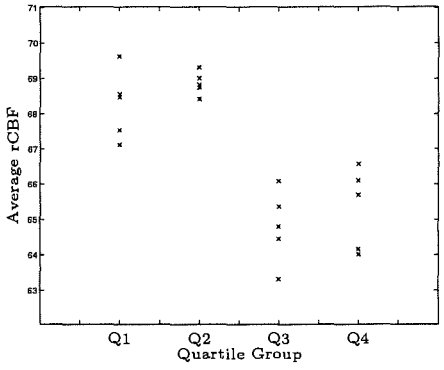


(a) Cluster N=139

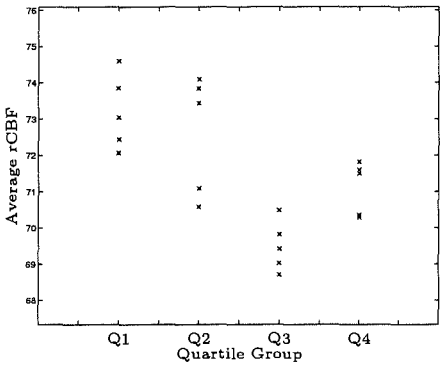
(b) Cluster N=478



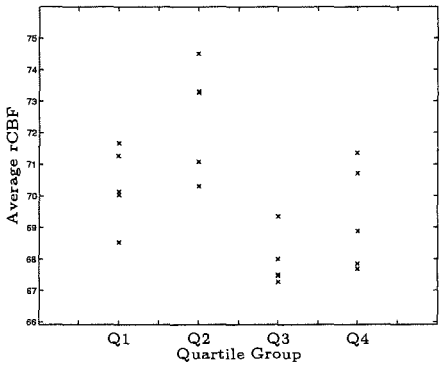
(c) Cluster N=503



(d) Cluster N=478, N=139 and N=503

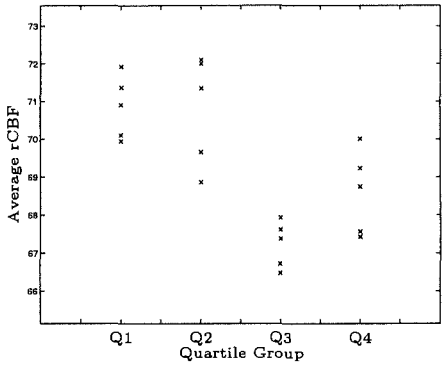


(e) Cluster N=112

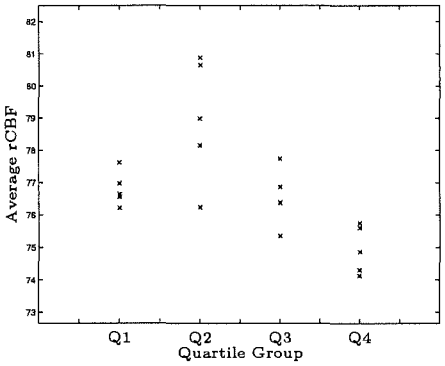


(f) Cluster N=258

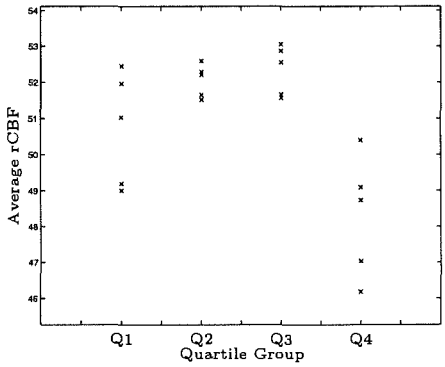
Figure 6.13: Average rCBF versus quartile group for cooperativeness.



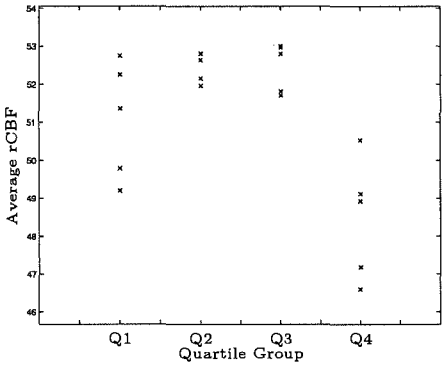
(a) Cluster N=258 and N=112



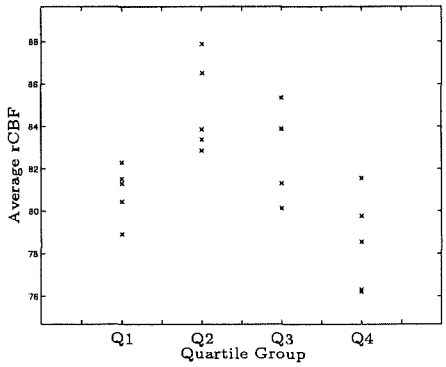
(b) Cluster N=128



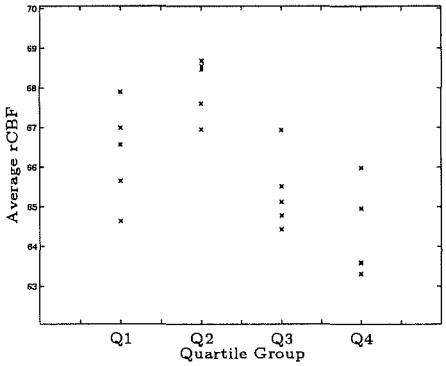
(c) Cluster N=93 (DQ3Q4)



(d) Cluster N=503 and N=93 (DQ3Q4)



(e) Cluster N=584



(f) Cluster N=100

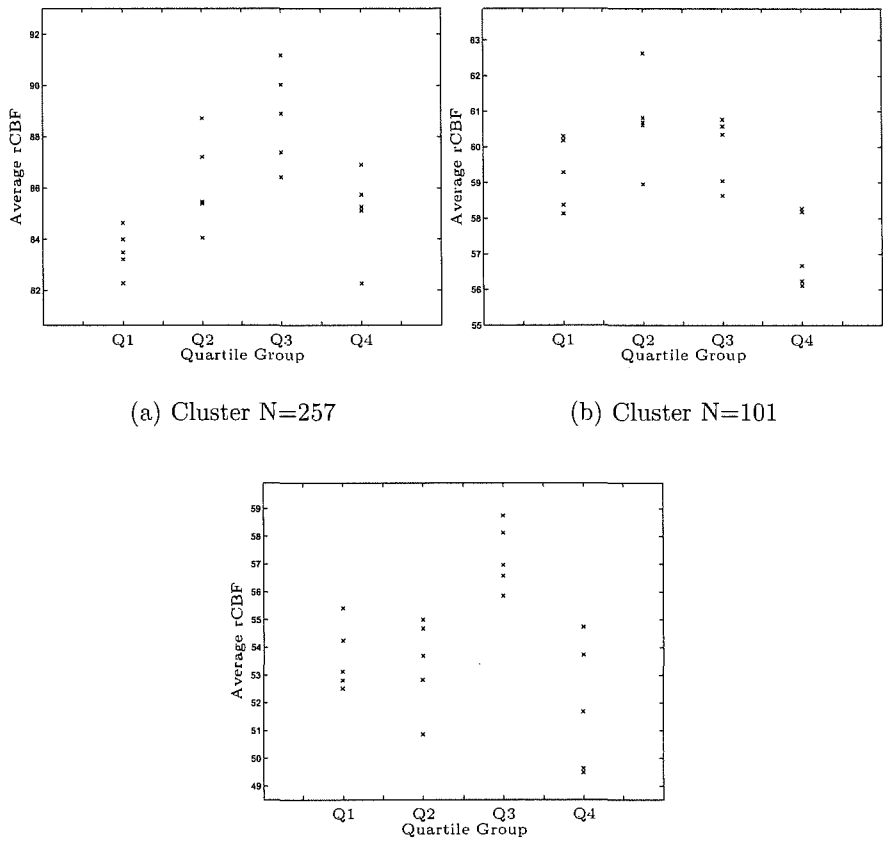
Figure 6.14: Average rCBF versus quartile group for cooperativeness.

6.5.7 Results for Self Transcendence

Three clusters were found to be related to differing levels of self transcendence. An activation was found (contrast Q1 and Q3) in the left occipital lobe encompassing the middle occipital gyrus, lingual gyrus, fusiform gyrus and calcarine. Figure 6.15(a) shows the general increase in rCBF from Q1 to Q3 with Q3 significantly higher than Q1. Q4 appears to be lower than Q3 but not significantly so. This indicates that rCBF increases with increasing self transcendence until a drop in rCBF for the highest levels.

The contrast between Q2 and Q4 found a small cluster of deactivated voxels ($N = 101$) located in the right middle and superior temporal gyrus. Figure 6.15(b) shows no change in rCBF from Q1 to Q3 and then a drop from Q3 to Q4, with Q2 significantly higher than Q4.

A large cluster of deactivation ($N = 446$) was found in the right superior temporal gyrus, inferior parietal gyrus, supra marginal gyrus and angular gyrus which was associated with a contrast between Q3 and Q4 (see Figure 6.15(c)). The cluster locations are shown in Figure 6.17(b).

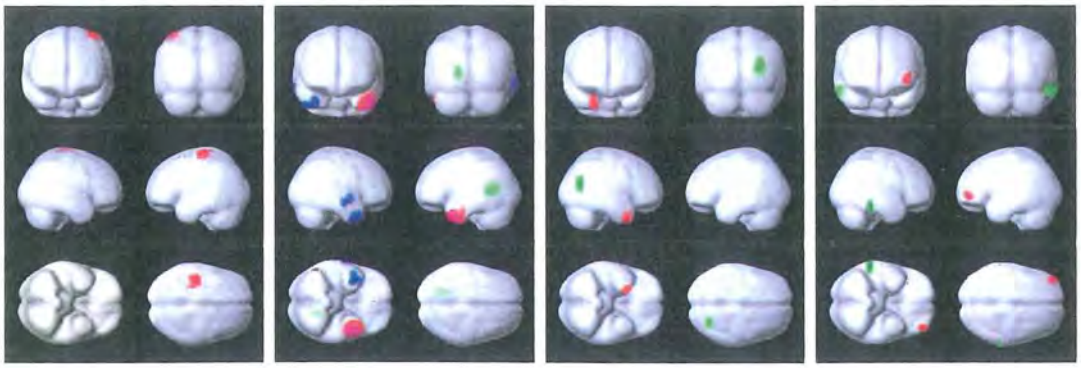


(a) Cluster N=257

(b) Cluster N=101

(c) Cluster N=446

Figure 6.15: Average rCBF versus quartile group for self transcendence.

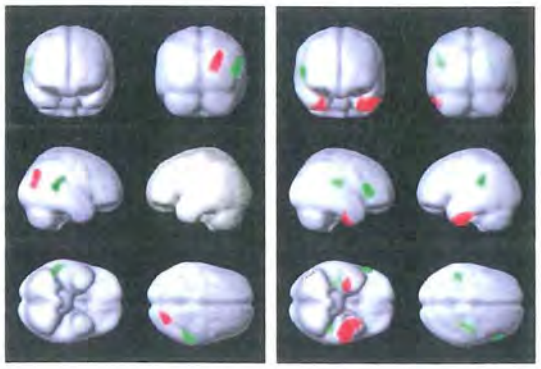


(a) NS: AQ2Q4 (337) = R

(b) NS: DQ1Q4 (1010) = R, DQ1Q3 (611) = G, DQ2Q4 (185,180,97) = B

(c) HA: AQ1Q3 (191) = R, AQ2Q3 (390) = G

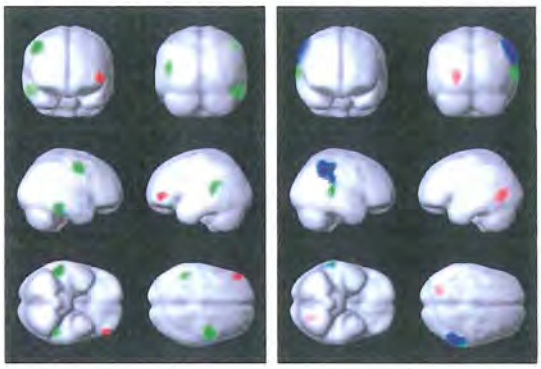
(d) RD: AQ1Q2 (118) = R, AQ1Q3 (163) = G



(e) RD: DQ2Q4 (227) = R, DQ3Q4 (131) = G

(f) P: AQ1Q3 (797,136) = R, DQ2Q3 (173,128,123) = G

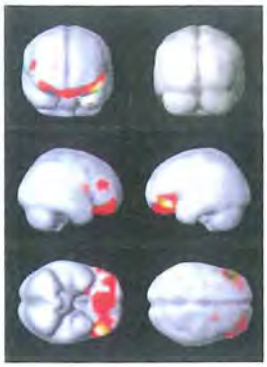
Figure 6.16: The temperament traits (Key: A = Activation, D = Deactivation, R = Red, G = Green, B = Blue).



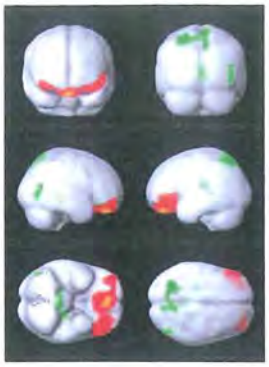
(a) S: AQ3Q4 (100) = R, DQ2Q3 (276,193,178) = G

(b) ST: AQ1Q3 (257) = R, DQ2Q4 (101) = G, DQ3Q4 (446) = B

Figure 6.17: The character traits, S and ST (Key: A = Activation, D = Deactivation, R = Red, G = Green, B = Blue).

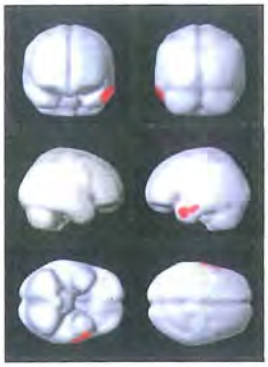


(a) C: AQ1Q2 (3108,93,92) = R, AQ1Q3 (470) = G, AQ1Q4 (115) = B

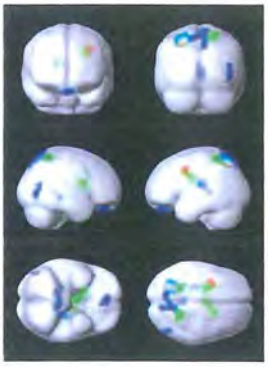


(b) C: AQ1Q2 (3108) = R, DQ2Q4 (584,503,128,103,100) = G

(c) C: DQ2Q3 (478,333) = R, AQ1Q2 (3108,93,92) = G



(d) C: AQ2Q3 (253) = R



(e) C: DQ1Q3 (139,112) = R, DQ2Q3 (478,333,258,153) = G, DQ2Q4 (584,503,128,103,100) = B

(f) C: DQ2Q4 (584,503) = R, DQ3Q4 (93) = G

Figure 6.18: The character traits, C (Key: A = Activation, D = Deactivation, R = Red, G = Green, B = Blue).

Self Transcendence	Cluster		
(Expected voxels per cluster N_E = 6.339)	Coordinates	N	p-value
Activation Q1 Q3			
Left Occipital Lobe:	(-22, -72, 0)	257	**0.000**
Middle occipital gyrus			
Lingual gyrus, Fusiform gyrus			
Calcarine			
Deactivation Q2 Q4			
Right Temporal Lobe:	(62, -38, 6)	101	0.040
Middle, superior temporal gyrus			
Deactivation Q3 Q4			
Right Temporal Lobe:	(54, -48, 46)	446	**0.000**
Superior temporal gyrus			
Right Parietal Lobe:			
Inferior parietal gyrus			
Supramarginal gyrus, Angular gyrus			

Table 6.9: Contrast results for self transcendence.

6.6 Summary

We believe this study is the first to investigate all seven of the TCI personality traits in relationship to rCBF. This study has some advantages; the age range of the participants is small (20 to 33), the participants are all male to avoid a gender effect, and the injection process was standardised so that all participants were completing the TCI questionnaire at the time of injection. Some studies use an eyes closed protocol, which leads to difficulties in assessing what patients were thinking about as they were injected.

The results show that using a quartile modelling approach with t-contrasts allowed for the detection of non-linear relationships between the four quartile levels, these would not have been detected with the standard modelling approach. The graphs of quartile grouping versus the average rCBF in the cluster indicated the generally non-linear relationship.

Sugiura et al. (2000) studies novelty seeking, harm avoidance and reward dependence in relationship to rCBF using a correlation approach within the original SPM96 program. Their study had some disadvantages. Whilst their sample size was ten larger than ours, they used both males and females but did not appear to have investigated a gender

confound effect. Sugiura et al. (2000) also had a large age range (26-61 years). They did check for brain atrophy and excluded those below a threshold value. The Sugiura et al. (2000) work also used an eyes closed injection protocol which leads to difficulties in assessing differences in thought patterns at the time of injection. What the person is thinking about at the time of tracer injection can have a big impact on the distribution of blood flow within the brain.

We believe the study protocol presented in this chapter leads to less confounding effects compared to the Sugiura et al. (2000) study. This combined with the novel quartile contrast approach implemented in this chapter has extended this type of study. In the following sections the results for each trait are presented in the context of the current literature in this field.

Novelty seeking was found to relate to an activation in the precentral and post central gyri, which correspond to the motor cortex and the somatic sensory cortex. Deactivations were found in the temporal gyri, occipital lobe and precuneus. The temporal lobe is involved with emotion and the precuneus is involved in the sensory association cortex. Novelty seeking is expected to be related to the amygdaloid subdivision of the brain (Cloninger, 2002), which influences drive related behaviour patterns and the corresponding subjective feelings (Nolte, 1981). Sugiura et al. (2000) found positive correlations between novelty seeking and the orbital prefrontal network. This network sends information to the amygaloid subdivision.

Increased levels of harm avoidance exhibited activations in the temporal and occipital lobes. Sugiura et al. (2000) showed a negative correlation between harm avoidance and the medial prefrontal network. Two areas of activation were found in the same regions as in Sugiura et al. (2000), namely in the right fusiform gyrus and parahippocampal gyrus. However Sugiura et al. (2000) showed these regions to be associated with deactivations rather than activations as shown by the quartile contrast approach in this study (Turner et al., 2003). This is not necessarily contradictory as the graphs in Figure 6.7 showed non-linear relationships between quartile personality groupings and average rCBF in the cluster. In both clusters, found in this study, there appears to be a deactivation trend also ($N = 191$ Q3 to Q4, $N = 390$ Q1 to Q2), but not significantly so.

Reward dependence is expected to be related to the circuit of Papez (Cloninger, 2002), which loops from the hippocampal formation, through the fornix, mammillary body, anterior thalamic nucleus, cingulate gyrus, part of the parahippocampal gyrus and back to the hippocampal formation (Nolte, 1981). This circuit involves the thalamo-cingulate subdivision of the limbic system. Sugiura et al. (2000) showed a relationship between reward dependence and a decrease in blood flow in the paralimbic regions. The work presented in this study, showed increased reward dependence related to activations in the frontal and temporal lobes and deactivations in the temporal and occipital lobes. An activation

was found in the left middle frontal gyrus in the same region as a negative correlation reported by Sugiura et al. (2000).

This, as far as we are aware, is the first functional brain imaging study to investigate the dependence of regional cerebral blood flow on persistence, self directedness, cooperativeness and self transcendence. Persistence, like reward dependence, exhibited activations in the temporal lobe, but unlike reward dependence had activations in the parietal, occipital and limbic lobes. Deactivations were found in the parietal, temporal and frontal lobes as well as the rolandic operculum and insula.

The character traits are expected to be related to higher cognitive functions (Cloninger, 2002). The brain regions thus expected to be related to the character traits are the thalamo-neocortical system and the prefrontal cortex (Cloninger, 2002). Self directedness showed activations in the frontal lobe only, with deactivations in the precentral gyrus, frontal lobe, temporal lobe and occipital lobe. Cooperativeness showed the most number of clusters and was also related to the largest cluster involving more than 3000 voxels. The activations were again strongly in the frontal lobes but also involved the limbic lobe, temporal lobe and sub cortical gray nuclei. Deactivations were found mainly in the parietal lobe, central region and occipital lobe with involvement also from the temporal lobe, middle frontal gyrus, subcortical gray nuclei, cerebellum and vermis. Self transcendence showed activations in the occipital lobe and optic radiation and deactivations in the temporal and parietal lobe. Further work on a larger sample of normal males is needed to confirm these relationships.

This chapter has presented the results from the investigation of the relationship between brain function and TCI personality traits in twenty normal males. These results are published in Turner et al. (2003). The results show a significant link between regional cerebral blood flow and the Cloninger personality model. This, as far as we are aware, is the first study to show a significant relationship in specific regions of the brain to persistence, self directedness, cooperativeness and self transcendence. These results support previous work showing a biological basis for the TCI model (Cloninger, 2002).

There are many aspects to this study that can be extended to enable further investigation of the links found. Ideally a multisubject brain atlas would be used for cluster location and when such an atlas is available it will be of interest to see what sort of affect, if any, this has on the cluster locations reported. The graphs presented in this chapter, showing the relationship between quartile personality groups and rCBF, are clearly non-linear in nature. This can be further investigate using the general additive models (GAMs) approach (this approach was used in Chapter 5 to investigate the relationship between personality and symptoms of depression). GAM modelling would replace the general linear model currently used and would model the non-linearity without having to prespecify the type.

A further extension to this work is to use non-parametric statistics such as permutation test. Permutation tests are available within the package SnPM (Nichols and Holmes, 2001), which is a tool box for SPM99. Other non-parametric tests could also be investigated. For example a concordance test could be used to investigate the concordance of the voxel responses in a particular region across the 20 subjects.

Chapter 7

Conclusion

This thesis investigated personality in two areas. The first area involved the investigation of the structure of personality and that of symptoms of depression, and the interrelationship between the resultant structures. The second part of the personality study investigated the relationship between brain function and personality types in normal males. Results from the Flury test showed that there were significant, or very nearly significant, gender differences on all but the baseline symptoms. This work plays an important role in current research by developing structural personality and symptom models and using these models to investigate changes in symptoms and personality with treatment for depression. This study uses independent component analysis, rather than principal component analysis or factor analysis with personality and symptom data.

The non-linear modelling technique of general additive models was used to thoroughly investigate the relationship between symptoms and personality. Finally all seven of the Cloninger TCI traits were investigated in relationship to brain function measured by SPECT (Turner et al., 2003). This supports previous work showing a biological basis for the Cloninger model.

This study is different to most of the studies in the literature to date, which investigate the factor structure of the questions rather than the Cloninger traits. This study had a relatively small sample size making analysis of the original questionnaire data unadvisable. The trait and symptom structures from the questions have been well documented in the literature and in this study have been used directly to investigate a possible reduced dimensionality for the latent structure.

7.1 Initial Statistical Analyses

Chapter 2 detailed the specific data collection protocols used in the study. Basic descriptive statistical analyses were conducted and showed that, in general, the data is not

normal and is often highly skewed. The symptom data is also often left truncated. The tests of hypotheses conducted demonstrated that there are significant changes before and after treatment in both the personality and symptoms of the depressed patients. Not all of the personality variables changed, but all of the symptoms improved.

Non-parametric hypothesis tests were used to compare the exploratory and confirmatory depressed patient datasets. This comparison of the exploratory and confirmatory data showed that at both time points there were few significant differences except for the baseline symptoms, which showed significant differences on most symptom variables. An area for future work is to investigate some of these differences in more detail. The data after treatment could be further investigated for differences due to the type of treatment regime. To investigate this properly a much larger sample would be needed which is not currently available for the personality data.

The three normal datasets were compared in a similar manner. The brain study normal males were significantly different to the never ill relatives of bipolar patients on novelty seeking, harm avoidance, self directedness and cooperativeness. As well as these differences the brain study males were significantly different to the never ill males on persistence and self transcendence. There were also significant differences between the never ill males and females on the traits harm avoidance, reward dependence and persistence. So the normal males for the brain study are not as "normal" as one would like but given that the type of study and recruitment were expected to add bias in the types of personality recruited, this is perhaps to be expected.

Gender differences were investigated in the depressed datasets. At both time points, and for both the exploratory and confirmatory datasets, males and females were significantly different on the personality traits of reward dependence and cooperativeness. No significant differences were found for novelty seeking, harm avoidance, persistence, self directedness and self transcendence. Investigation of gender differences in the depression symptoms of the exploratory dataset found that at baseline the only significant difference was for the symptom of paranoid ideation. After treatment however, five symptoms are significantly different, namely obsessive compulsive symptoms, interpersonal sensitivity, depression, anxiety and paranoid ideation. The confirmatory dataset had no significant gender differences at baseline and two significant differences after treatment, namely obsessive compulsive symptoms and psychotocism.

The changes in symptoms and personality across time were investigated. The exploratory females had significant differences across time for harm avoidance, reward dependence, self directedness and cooperativeness. The confirmatory females had significant differences in harm avoidance, persistence, self directedness and cooperativeness. For the exploratory males, novelty seeking, harm avoidance, self directedness and cooperativeness were significantly different across time. The confirmatory males were significantly differ-

ent across time in novelty seeking, harm avoidance, reward dependence and self directness. Two traits were consistently different across time, in all four groups, namely harm avoidance and self directedness. The symptoms were all significantly different across time for all four groups, as would be expected as the symptoms are improving with treatment.

One of the main motivations of the bootstrapping in the structural equation modelling and for the quartile approach to the brain modelling was the inherent non-normality of the data.

7.2 Exploratory Component Analysis

Chapter 3 used the novel independent component approach, which subsumes principal component analysis and factor analysis, to develop models that described the underlying personality and symptom structure of the exploratory dataset. Whilst many studies on personality and symptoms of depression have used principal component or factor methods to analyse this type of data, none, as far as the author is aware, have used the recent advance of independent component analysis.

The models presented in Chapter 3 and Appendix A showed that the three component methods lead, at times, to quite different solutions. The independent component method produced some symptom factors that were contrasts between the symptom variables. Principal component analysis and factor analysis both produced only positive loadings for the symptom variables on the underlying factors. The symptoms are expected to be positively correlated as a person suffering under psychological distress will score highly on all the symptoms, compared to the normal population. For the personality data the three methods produced similar results.

The Flury test was used initially to determine if there were significant differences between males and females on personality and symptoms. The results showed that there were significant differences in the after treatment symptoms, but not so for baseline. Not only were three different methods used to investigate the component or factor structure, different methods were used to calculate the number of components to retain. These methods were compared after confirmatory factor analysis on a second confirmatory dataset, adding rigour to the analyses.

This study is different to most of the studies in the literature to date, which investigate the factor structure of the questions rather than the traits. The trait and symptom structures from the questions have been well documented in the literature and in this study have been used directly to investigate the underlying structure, in that, the study is investigating how the traits interact.

7.3 Confirmatory Factor Analysis, Multigroup Analysis and Longitudinal Modelling

Using confirmatory factor analysis (CFA) Chapter 4 found the best structural model from those developed in Chapter 3, on the second dataset introduced in Chapter 2. The models were bootstrapped to counter normality issues and provide confidence intervals for both the parameters and for the fit indices.

There were eight final models in total and five of these models resulted from the independent component analysis. Thus ICA was the best method to use; not surprising as ICA takes component analysis to the next level by striving for independence of components, rather than just decorrelated, as is the case in PCA. As far as the author is aware no other studies have investigated TCI components using the seven traits rather than the original questions. In all cases the TCI traits were reduced to one component for both genders at most time points.

There is debate as to how many components the SCL questionnaire actually measures. This study has found that, when the depressed patients are severely depressed, six factors are needed to describe the symptoms adequately. However after treatment, when symptoms have reduced substantially, only two factors (for the females) are needed. This, in part, explains the differences seen in the literature for the number factors the SCL questionnaire measures. Studies with severely ill patients may find more factors than those with normal or less severely ill patients.

Some studies, such as those by Carpenter and Hittner (1995), Bonynge (1993) and Bernstein et al. (1994), have shown evidence of a single overall symptom factor of general distress, rather than nine distinct symptoms as presented by Derogatis and Cleary (1977). However Bernstein et al. (1994) suggested a second factor may be appropriate, separate from the overall distress measure. Steer et al. (1994) not only found an overall general component of distress but also identified four specific residual components that were appropriate for their study. Studies such as those by Vassend and Skrondal (1999) and Schwarzwald et al. (1991) presented evidence for more than one factor.

A second point of contention with the symptom checklist is about the presence or absence of gender differences in the symptom structure. The Flury test from Chapter 3, showed significant, or nearly significant gender differences for personality at both time points and for the symptoms after treatment. The baseline symptoms were found to have common gender components. Multigroup analysis further investigated these results and generally there were significant gender differences. Bonynge (1993) showed gender invariance in a group of suicidal adults and adolescents. Vassend and Skrondal (1999); Carpenter and Hittner (1995) both showed significant gender differences, on data from, in the first case, the general population and data from psychiatric patients.

The combined symptom and personality models were interesting. The final models either had no personality variables or very few of them and were driven basically by symptoms. This suggests that when personality and symptom variables are combined in the analysis the symptoms dominate. There are few relationships between the symptom and personality variables in the exploratory and confirmatory factor analysis environment.

The longitudinal analysis showed that for the females, harm avoidance, persistence, and self directedness were important at both time points and maintained similar loadings. Cooperativeness was important at baseline but was replaced by self transcendence after treatment. The underlying personality factor was significantly correlated across time suggesting similar patterns of these personality traits across time. The factor scores were stable across time. In comparison the male's personality factor scores were not stable across time.

The symptoms at baseline had six underlying factors and after treatment two factors. The factors were highly correlated across time suggesting those with comparatively high symptoms at baseline tended to have comparatively high symptoms after treatment. The factor loadings after treatment are smaller, showing an improvement of symptoms across time.

The biggest drawback to the analyses was the small sample size, particularly for the males. This could explain why, in some instances, no adequate male personality or symptom model was found. The confirmatory factor analysis technique allows variables to only load on to one underlying factor. This is an area that can be extended in future work by allowing the variables to cross load onto the factors.

Discriminant analysis and logistic regression were used to investigate the interpretability of the components. This analysis found that, for the males, the first symptom component has a protective effect in that higher levels predict greater improvement. The second symptom component was a risk factor for poor improvement, in that high levels predicted and discriminated poor improvement as measured by the change in Hamilton score.

7.4 Predicting Symptoms of Depression from Personality

The analysis of the relationship between personality and symptoms of depression is presented in Chapter 5. To thoroughly investigate any potential relationship between symptoms and personality general additive models (GAMS) were used. These models allow for non-linearity without prespecifying the type of non-linearity. This chapter extends the work in the literature by using non-linear modelling, treating males and females separately, and by investigating not only the prediction of symptoms from personality but

also the prediction of personality from the symptoms of depression. Predicting symptoms from personality produced better fit models. This supports the directionality usually seen in the literature.

The character personality traits were particularly important at baseline. After treatment the temperament traits became more significant. After treatment there appears to be more of a link between personality and symptoms. This thesis extended the work of Grucza et al. (2003) by investigating all seven of the personality traits.

7.5 Personality as a Predictor of Brain Function

We believe this study is the first to investigate all seven of the TCI personality traits in relationship to regional cerebral blood flow (rCBF). The results show the value of using a novel quartile approach with t-contrasts to modelling personality as a predictor of rCBF. The results were compelling, as many rCBF clusters were significantly related to personality when using a conservative Bonferroni adjustment for the multiple quartile comparisons. The study also clearly showed the non-linear nature of the relationship. The results support the idea of a biological basis for the Cloninger personality model.

In this study novelty seeking was found to relate to an activation in the precentral and post central gyri, which correspond to the motor cortex and the somatic sensory cortex. Deactivations were found in the temporal gyri, occipital lobe and precuneus. Increased levels of harm avoidance exhibited activations in the temporal and occipital lobes in this study. Reward dependence was expected to be related to the circuit of Papez (Cloninger, 2002), which loops from the hippocampal formation, through the fornix, mammillary body, anterior thalamic nucleus, cingulate gyrus, part of the parahippocampal gyrus and back to the hippocampal formation (Nolte, 1981). The work presented in this chapter showed increased reward dependence related to activations in the frontal and temporal lobes; and increased reward dependence was associated with deactivations in the temporal and occipital lobes. An activation was found in the left middle frontal gyrus in the same region as a negative correlation reported by Sugiura et al. (2000). Persistence, like reward dependence, exhibited activations in the temporal lobe, but unlike reward dependence had activations in the parietal, occipital and limbic lobes. Deactivations were found in the parietal, temporal and frontal lobes as well as the rolandic operculum and insula.

The brain regions expected to be related to the character traits, are the thalamo-neocortical system and the prefrontal cortex (Cloninger, 2002). In this study we showed that self directedness was related to activations in the frontal lobe only, with deactivations in the precentral gyrus, frontal lobe, temporal lobe and occipital lobe. Cooperativeness showed the most number of clusters and was also related to the largest cluster involving more than 3000 voxels. These activations were again strongly located in the frontal lobes

but also involved the limbic lobe, temporal lobe and sub cortical gray nuclei. Deactivations associated with cooperativeness, were found mainly in the parietal lobe, central region and occipital lobe with involvement also from the temporal lobe, middle frontal gyrus, subcortical gray nuclei, cerebellum and vermis. Self transcendence showed activations in the occipital lobe and optic radiation and deactivations in the temporal and parietal lobe.

7.6 Overview of the Study

This is the first study, as far as the author is aware, to investigate the relationship between personality and symptoms of depression using latent structure modelling and general additive models. The latent structure modelling was comprehensively conducted with cross validation using three different methods for model development i.e. PCA, ICA and FA. This is the first study to apply independent component analysis to psychometric data and the independent component models were the best confirmed models in five of the eight final models. This study also used the latent structure modelling to investigate the personality and symptoms across time. The models developed found that personality could be described by one component rather than the original seven traits and that the symptoms could be described by six components before treatment and only two components after treatment instead of the original nine symptoms.

The general additive models showed that at baseline the character personality traits were important in the prediction of depression symptoms. Generally the literature has concentrated on the temperament traits. The use of the character traits allows for more knowledge to be gained into the relationship between depression and personality.

The brain study has some major advantages in that the age range of the participants was small (20 to 33 years), the participants were all male to avoid a gender effect, and the injection process was standardised so that all participants were completing the TCI questionnaire at the time of injection. Some studies use an eyes closed protocol, which leads to difficulties in assessing what patients were thinking about as they were injected. The sample size of twenty is too small for results to be generalised to a wider population and future studies are needed to draw conclusive results about the relationship between personality and regional cerebral blood flow. The study does show that even in a small sample, interesting and significant results can be gained. This study also shows that quartiles, used as conditions in the modelling technique, accommodates for any non-linear relationship between personality and rCBF.

7.7 Future Work

There are a number of areas that can be further investigated to build on the work presented in this thesis. One such area is to extend the confirmatory factor analysis to allow more complicated models. The confirmatory analyses could be extended by increasing the sample size. A larger sample size would allow the use of more complicated modelling, and also the use of better estimation techniques that are more suitable for the non-normal data. This type of analysis has applications to randomised control studies. Components can be developed that better describe and predict treatment outcomes.

There are many avenues for further work on the brain study. Non-linear modelling is an obvious place to start as the graphs of the personality quartiles versus the average brain blood flow in a cluster show non-linear relationships. The non-linear modelling would be hard to implement in a computational sense and the programming as such may be intensive. Other currently available methods for analysing the brain images, are non-parametric permutation tests, which are suited to the studies with low degrees of freedom and principal components of brain images. Another area to extend the brain work is in the cluster location. Currently the author was only able to use atlases based on single subjects. Ideally an atlas based on many subjects would be used.

Other areas to extend the brain analysis are the use of independent component analysis on the brain images to reduce the dimensionality. This could form the basis of structural equation modelling between brain regions and personality. Bayesian analysis methods have recently been developed for functional magnetic resonance imaging and could be extended to the SPECT images.

Appendix A

Component Analysis Models

This chapter presents all the solutions found in Chapter 3, using principal components analysis, factor analysis and independent components analysis for varying numbers of components retained. These models are based on the exploratory dataset and are tested in a confirmatory factor analysis framework in Chapter 4 using the confirmatory dataset. These methods have reduced the dimensionality of the personality and symptom data.

A.1 The Depressed Females Personality

Baseline Results

Table A.1 presents information on the eigenvalues and variance accounted for the depressed females at baseline. The first component accounts for 36.5% of the variance and five components are needed to account for more than 90% of the variance (percentage variance accounted for method discussed in Section 3.6.5).

The suggested number of components to retain for the depressed females at baseline are 1,3,4 and 5. These component solutions are presented in Tables A.2 to A.5. The one component solutions are presented in Table A.2. The PC and IC methods lead to

	Eigenvalue	Proportion	Cumulative
1	0.094	0.365	0.365
2	0.056	0.218	0.583
3	0.037	0.145	0.728
4	0.035	0.135	0.862
5	0.019	0.074	0.936
6	0.010	0.038	0.974
7	0.007	0.026	1.000

Table A.1: Eigenvalues and variance for the personality of the depressed females at baseline.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	**-0.396**	**0.704**	-0.093
Harm Avoidance	**-0.449**	**0.909**	**-0.550**
Reward Dependence	0.082	-0.152	0.021
Persistence	**0.929**	**-2.790**	**0.213**
Self Directedness	**0.530**	**-1.154**	**1.000**
Cooperativeness	0.308	-0.428	**0.439**
Self Transcendence	0.169	-0.293	0.004

Table A.2: Depressed females TCI at baseline, one component solution.

TCI Traits	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
Novelty Seeking	-0.324	-0.312	**-0.623**	**-1.983**	-1.382	-0.277	-0.139	-0.121	**0.983**
Harm Avoidance	**0.923**	0.026	0.053	**2.850**	0.459	-1.110	**0.982**	0.112	-0.153
Reward Dependence	0.147	**0.708**	0.033	**2.502**	0.027	2.249	-0.001	**0.454**	0.057
Persistence	-0.298	-0.032	**0.943**	-0.826	**3.218**	-0.631	-0.297	0.012	**-0.439**
Self Directedness	**-0.730**	0.520	0.047	-0.503	-0.246	**3.069**	**-0.619**	0.325	-0.143
Cooperativeness	-0.095	**0.802**	0.128	1.625	0.106	**2.098**	-0.164	**0.975**	-0.152
Self Transcendence	-0.282	-0.069	0.037	-0.894	-0.003	0.060	-0.173	0.117	0.205

Table A.3: Depressed females TCI at baseline, three component solution.

the same model with novelty seeking and harm avoidance in contrast with persistence and self directedness. The factor analysis model (FA 1) contrasts harm avoidance against persistence, self directedness and cooperativeness. Even though persistence has a small loading it was retained so that the model was uniquely identified.

Table A.3 presents the three component solutions. The PC and FA solutions lead to similar conclusions, whilst the IC solution is a different combination. The first PC measures harm avoidance versus self directedness; persistence is now factored out into the third PC in contrast to novelty seeking. The second PC measures reward dependence, cooperativeness and self directedness. The first IC however, measures harm avoidance and reward dependence. The second IC measures persistence and appears similar to the third PC (PC 3). The third IC measures self directedness, reward dependence and cooperativeness.

The four component solutions are different across the three methods. The four component solution is shown in Table A.4. The first PC remains similar to the first PC in the three factor solution, namely self directedness versus harm avoidance. The second PC is also similar (RD and C) but self directedness does not feature as strongly. The third PC measures self transcendence and novelty seeking versus harm avoidance. The fourth PC is similar to the third PC of the 3 component solution and measures persistence versus novelty seeking. The IC solution is also shown in Table A.4. The first IC measures reward dependence versus self directedness. The second IC is a persistence component. Novelty seeking and self transcendence are contrasted with harm avoidance to form the third IC (IC 3). The last IC (IC 4) measures a combination of reward dependence, self directedness and cooperativeness.

TCI Traits	PC 1	PC 2	PC 3	PC 4	IC 1	IC 2	IC 3	IC 4
Novelty Seeking	0.125	-0.213	**0.671**	-0.513	0.456	0.893	**-2.998**	-0.424
Harm Avoidance	**-0.815**	0.136	-0.412	-0.083	1.685	-0.254	**2.494**	-0.738
Reward Dependence	-0.125	**0.882**	0.238	0.003	**3.469**	-0.465	-0.087	2.697
Persistence	0.153	0.020	0.066	**0.980**	-0.249	**-3.325**	-0.554	-0.743
Self Directedness	**0.911**	0.248	-0.189	0.083	-2.254	0.573	0.708	**2.874**
Cooperativeness	0.272	**0.754**	-0.152	0.084	0.953	-0.099	0.942	**2.304**
Self Transcendence	0.004	0.164	**0.749**	0.136	1.959	-0.661	**-2.834**	0.101
TCI Traits	FA 1	FA 2	FA 3	FA 4				
Novelty Seeking	0.111	-0.154	**0.802**	0.567				
Harm Avoidance	**-0.672**	0.122	0.029	-0.402				
Reward Dependence	-0.063	**0.474**	-0.004	0.149				
Persistence	0.172	0.017	**-0.671**	0.230				
Self Directedness	**0.925**	0.206	-0.117	-0.124				
Cooperativeness	0.239	**0.962**	-0.120	-0.046				
Self Transcendence	0.032	0.125	-0.034	**0.449**				

Table A.4: Depressed females TCI at baseline, four component solution.

TCI Traits	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Novelty Seeking	-0.097	**0.837**	0.003	-0.314	0.164	-1.447	**2.333**	2.099	0.393	-1.661
Harm Avoidance	-0.620	**-0.657**	0.074	-0.271	-0.065	0.372	**-2.763**	-1.761	1.600	0.571
Reward Dependence	-0.031	0.047	**0.985**	0.030	0.029	-0.755	-1.785	**5.029**	0.796	0.348
Persistence	0.117	-0.143	0.023	**0.976**	0.083	**3.131**	0.466	1.164	0.169	-1.356
Self Directedness	**0.971**	-0.020	0.040	0.089	-0.022	0.011	0.389	-0.782	-2.163	**3.313**
Cooperativeness	0.492	-0.380	**0.554**	-0.028	0.170	0.036	-1.187	0.210	0.550	**2.779**
Self Transcendence	0.017	0.130	0.070	0.075	**0.980**	0.513	2.163	-1.960	**4.818**	2.850

Table A.5: Depressed females TCI at baseline, five component solution.

Factor analysis was not possible for a five component solution, from SAS's (SAS(R) Proprietary Software Release (8.1)) inbuilt criteria, so only the PC and IC solutions are presented. The five component solution loadings are shown Table A.5. The first PC is similar to the first PC in the three and four component solutions; i.e. harm avoidance versus self directedness. In this solution cooperativeness is a stronger component. The third PC in the four factor solution appears to have split into PC2 (NS versus HA) and PC5 (ST). PC3, in the 5 component solution, is similar to PC2 in the 4 component solution and PC4 is similar to PC4 in the 4 component solution, with novelty seeking less important. The first IC measures persistence (similar to PC4), the second IC measures harm avoidance versus novelty seeking and self transcendence; reward dependence and novelty seeking are measured by the third IC. The fourth IC is essentially self transcendence and the fifth IC is essentially self directedness and cooperativeness.

Post Treatment Results

Table A.6 presents the eigenvalues and variance for the personality of the depressed females at six months. In comparison to the values at baseline (Table A.1) the variance accounted for by each component has changed little and five components are required to

	Eigenvalue	Proportion	Cumulative
1	0.083	0.342	0.342
2	0.055	0.225	0.568
3	0.040	0.163	0.730
4	0.028	0.114	0.844
5	0.022	0.089	0.933
6	0.010	0.040	0.973
7	0.007	0.027	1.000

Table A.6: Eigenvalues and variance for the personality of the depressed females at six months.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	-0.044	-0.083	0.082
Harm Avoidance	**-0.597**	**-1.352**	**0.578**
Reward Dependence	0.035	0.068	0.058
Persistence	**0.866**	**2.817**	**-0.159**
Self Directedness	**0.581**	**1.450**	**-1.000**
Cooperativeness	**0.253**	0.351	**-0.435**
Self Transcendence	0.196	**0.405**	-0.003

Table A.7: Depressed females TCI at six months, one component solution.

retain at least 90% of the variance.

The one component solution for the depressed females at six months is similar to the one component solution at baseline. This component measures persistence, self directedness and cooperativeness versus harm avoidance (Table A.7) for both the PC and FA solutions. The IC solution differs with the inclusion of self transcendence rather than cooperativeness.

The first principal component of the three component solution (Table A.8) measures self directedness and cooperativeness versus harm avoidance. The second principal component measures novelty seeking, self transcendence and reward dependence and the third persistence. The first IC in the three component solution is essentially persistence (similar to PC3); the second independent component contrasts self directedness and cooperativeness against persistence; and the third measures self transcendence and novelty seeking versus harm avoidance. The first factor contrasts harm avoidance and self directedness, the second is a weighted average of reward dependence and cooperativeness, and the third

TCI Traits	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
Novelty Seeking	0.133	**0.737**	-0.211	0.733	-1.489	**2.451**	0.036	-0.042	**0.835**
Harm Avoidance	**-0.831**	-0.255	-0.106	-0.210	-1.307	**-2.270**	**0.879**	0.053	-0.419
Reward Dependence	-0.047	**0.446**	0.044	0.007	**-1.304**	1.313	0.073	**0.340**	0.179
Persistence	0.123	0.016	**0.990**	**-3.705**	-0.443	0.014	-0.283	-0.035	-0.095
Self Directedness	**0.925**	-0.216	0.053	0.264	**3.326**	0.812	**-0.707**	0.264	-0.070
Cooperativeness	**0.398**	0.047	0.016	0.104	**0.537**	0.505	-0.246	**0.964**	-0.103
Self Transcendence	0.089	**0.766**	0.119	-0.015	-1.987	**2.629**	-0.001	0.245	**0.371**

Table A.8: Depressed females TCI at six months, three component solution.

TCI Traits	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Novelty Seeking	-0.080	**0.873**	0.076	0.205	-0.116	1.137	-1.525	-2.249	**2.427**	1.741
Harm Avoidance	**-0.692**	-0.608	0.049	0.020	-0.204	1.435	2.059	1.156	**-2.095**	-1.525
Reward Dependence	-0.029	0.085	**0.985**	0.042	0.024	0.142	**3.950**	1.616	3.333	2.390
Persistence	0.089	-0.055	0.015	0.059	**0.991**	0.825	-1.197	**2.000**	-1.270	**2.552**
Self Directedness	**0.969**	-0.065	-0.033	-0.008	0.063	**-2.728**	-0.544	1.821	0.674	-1.793
Cooperativeness	**0.510**	-0.325	0.413	0.296	-0.054	-0.194	1.249	**2.186**	0.770	-1.507
Self Transcendence	0.026	0.180	0.065	**0.970**	0.067	**3.669**	-1.784	1.971	0.961	-3.585

TCI Traits	FA 1	FA 2	FA 3	FA 4
Novelty Seeking	0.061	**0.986**	0.006	-0.153
Harm Avoidance	**-0.625**	-0.318	0.017	-0.211
Reward Dependence	-0.080	0.134	**0.369**	0.040
Persistence	0.122	-0.036	0.038	**0.719**
Self Directedness	**0.984**	-0.138	0.102	0.041
Cooperativeness	0.324	-0.171	**0.929**	-0.050
Self Transcendence	0.014	**0.330**	0.277	0.154

Table A.9: Depressed females TCI at six months, five component solution.

a weighted average of novelty seeking and self transcendence.

The five component solution using PCA, shown in Table A.9, has a first component that contrasts harm avoidance against self directedness and cooperativeness, the remaining four components have the single indicators of novelty seeking, reward dependence, self transcendence and persistence respectively. The first IC in the five component solution measures self transcendence versus self directedness. The second IC is a measure of reward dependence. A weighted average of cooperativeness and persistence is measured by the third IC. The fourth IC contrasts novelty seeking with harm avoidance. Persistence is measured by the fifth IC. By the SAS (SAS(R) Proprietary Software Release (8.1)) criteria only four factors were needed. The first factor contrasts harm avoidance against self directedness, the second is a weighted average of novelty seeking and self transcendence, the third a weighted average of reward dependence and cooperativeness. Persistence is measured by the fourth factor.

Comparison Across Time

For the female's personality at baseline, one, three, four and five component solutions were retained. After treatment only one component, three component and five component solutions were needed. Looking at the one component solution the PC and IC solutions are different across time, however the FA solution is the essentially the same at both time points (see Tables A.2 and A.7). A similar result is seen across time with the three component solution (Tables A.3 and A.8). However this time the FA solution has the same first two components across time. FA 3 contrasts novelty seeking against persistence at baseline, after treatment novelty seeking is contrasted against self transcendence. The five component solutions, presented in Tables A.5 and A.9, again show differences across time for both the PC and IC methods. Five components were too many for factor analysis, so

	Eigenvalue	Proportion	Cumulative
1	0.090	0.358	0.358
2	0.067	0.269	0.626
3	0.028	0.112	0.738
4	0.023	0.090	0.828
5	0.021	0.085	0.914
6	0.013	0.052	0.966
7	0.009	0.034	1.000

Table A.10: Eigenvalues and variance for the personality of the depressed males at baseline.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	**-0.549**	**-0.987**	**0.380**
Harm Avoidance	**0.754**	**1.939**	**-0.785**
Reward Dependence	-0.011	-0.016	0.113
Persistence	**0.634**	**1.848**	-0.161
Self Directedness	**-0.708**	**-1.456**	**0.789**
Cooperativeness	**-0.455**	-0.712	**0.583**
Self Transcendence	0.352	0.707	-0.089

Table A.11: Depressed males TCI at baseline, one component solution.

the four component solutions can be compared, and the factors are different across time (Tables A.4 and A.9).

A.2 The Depressed Males Personality

Baseline Results

The eigenvalues and proportion of the variance accounted for are presented in Table A.10 for the depressed males from the exploratory dataset. Five components are required to retain at least 90% of the variance.

At baseline the one component PC solution contrasts harm avoidance and persistence against self directedness, novelty seeking and cooperativeness. The IC solution measures harm avoidance and persistence versus self directedness (Table A.11). The factor solution contrasts harm avoidance versus novelty seeking, self directedness and cooperativeness.

TCI Traits	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
Novelty Seeking	**0.474**	-0.280	-0.498	**0.856**	0.011	**-0.516**
Harm Avoidance	**-0.935**	-0.037	-0.450	**-2.679**	-0.383	**0.724**
Reward Dependence	0.131	0.174	0.364	0.274	**0.395**	0.160
Persistence	-0.114	**0.962**	**3.387**	0.111	0.030	0.247
Self Directedness	**0.815**	-0.059	0.081	**1.833**	0.486	**-0.572**
Cooperativeness	**0.604**	0.080	0.285	**1.068**	**0.876**	-0.141
Self Transcendence	-0.067	**0.529**	**1.283**	0.034	0.080	0.206

Table A.12: Depressed males TCI at baseline, two component solution

TCI Traits	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Novelty Seeking	0.091	0.077	**0.909**	-0.138	-0.100	-0.737	-0.699	-2.739	-1.979	**-4.151**
Harm Avoidance	**-0.810**	0.084	-0.501	-0.072	-0.008	-0.447	-0.842	-1.391	-0.339	**2.900**
Reward Dependence	0.019	**0.934**	0.086	0.060	0.065	1.100	0.307	**-5.492**	1.508	0.447
Persistence	-0.070	0.053	-0.126	**0.973**	0.161	0.400	**3.731**	-0.097	-1.603	-0.093
Self Directedness	**0.894**	0.029	0.012	-0.119	-0.026	1.568	0.620	1.935	**2.408**	0.850
Cooperativeness	**0.660**	0.532	-0.086	-0.030	-0.021	1.827	0.655	-1.602	**2.331**	1.370
Self Transcendence	-0.020	0.058	-0.095	0.157	**0.980**	**-4.906**	-0.231	0.007	3.188	-1.254

TCI Traits	FA 1	FA 2	FA 3	FA 4
Novelty Seeking	0.177	-0.219	0.039	**0.959**
Harm Avoidance	**-0.722**	0.018	0.043	-0.293
Reward Dependence	0.094	0.115	0.988	0.031
Persistence	-0.065	**0.998**	-0.012	-0.016
Self Directedness	**0.822**	-0.106	-0.027	0.064
Cooperativeness	**0.633**	0.006	0.284	-0.045
Self Transcendence	-0.017	**0.327**	0.077	-0.126

Table A.13: Depressed males TCI at baseline, five component solution,

The loadings for the two component solution for the males at baseline are shown in Table A.12. The first principal component contrasts harm avoidance against self directedness, cooperativeness and novelty seeking. The second principal component is a weighted average of persistence and self transcendence. The IC solution presents a similar structure. The first factor is a weighted mean of reward dependence and cooperativeness. The second factor is a contrast of novelty seeking and self directedness against harm avoidance.

The five component solution is shown in Table A.13. The first principal component measures self directedness and cooperativeness versus harm avoidance; the second reward dependence and the third novelty seeking. The fourth and fifth measure persistence and self transcendence respectively. The first component in the IC solution is essentially self transcendence, the second component persistence and the third component reward dependence. The fourth component is an average of self directedness and cooperativeness. The fifth component measures a contrast between novelty seeking and harm avoidance. For the factor analysis only four factors were retained. The first contrasting self directedness and cooperativeness versus harm avoidance; the second contrasting persistence and self transcendence. Reward dependence loaded highly on the third factor and novelty seeking loaded highly on the fourth factor.

Post Treatment Results

Six months later five components are required to retain at least 90% of the variance (see Table A.14). The one component solution for the depressed males contrasts harm avoidance against self directedness, persistence and cooperativeness (Table A.15) for all three methods.

The two component solutions are shown in Table A.16. The first PC contrasts harm avoidance versus self directedness and cooperativeness. The second component contrasts

	Eigenvalue	Proportion	Cumulative
1	0.118	0.422	0.422
2	0.065	0.232	0.654
3	0.035	0.125	0.778
4	0.026	0.094	0.872
5	0.017	0.062	0.934
6	0.011	0.039	0.973
7	0.008	0.027	1.000

Table A.14: Eigenvalues and variance for the personality of the depressed males at six months.

TCI Traits	PC 1	IC 1	FA 1
Novelty Seeking	0.105	0.146	0.001
Harm Avoidance	**-0.895**	**-2.067**	**-0.674**
Reward Dependence	0.314	0.350	0.245
Persistence	**0.609**	**1.265**	**0.346**
Self Directedness	**0.852**	**1.435**	**0.995**
Cooperativeness	**0.574**	**0.758**	**0.638**
Self Transcendence	-0.061	-0.108	-0.015

Table A.15: Depressed males TCI at six months, one component solution.

TCI Traits	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
Novelty Seeking	0.092	**-0.662**	**1.458**	0.817	0.048	**0.999**
Harm Avoidance	**-0.888**	0.352	-0.431	**-2.462**	**-0.711**	-0.284
Reward Dependence	0.313	-0.037	-0.087	0.344	0.275	0.081
Persistence	0.621	**0.637**	**-2.756**	0.146	**0.406**	-0.328
Self Directedness	**0.852**	-0.006	-0.619	**1.295**	**0.960**	-0.047
Cooperativeness	**0.577**	0.129	**-0.618**	0.553	**0.648**	-0.103
Self Transcendence	-0.047	**0.685**	**-1.967**	-1.004	-0.022	-0.195

Table A.16: Depressed males TCI at six months, two component solution.

TCI Traits	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
Novelty Seeking	0.241	**-0.653**	-0.192	**-1.728**	0.837	0.869	0.052	0.047	**0.672**
Harm Avoidance	**-0.914**	0.092	0.270	0.147	-1.431	**-2.104**	-0.343	**0.794**	-0.502
Reward Dependence	0.328	-0.001	0.011	-0.116	-0.029	0.477	**0.367**	-0.088	0.022
Persistence	0.419	**0.875**	-0.001	**3.819**	-0.500	-0.434	0.089	**-0.625**	-0.425
Self Directedness	**0.860**	0.116	0.023	-0.003	-0.148	**1.713**	0.576	**-0.579**	0.027
Cooperativeness	**0.624**	0.051	0.282	-0.594	-0.853	**1.429**	**0.976**	-0.135	-0.173
Self Transcendence	0.034	0.151	**0.952**	-1.810	**-3.832**	1.869	0.069	0.024	**-0.341**

Table A.17: Depressed males TCI at six months, three component solution.

TCI Traits	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Novelty Seeking	-0.019	**0.886**	0.139	-0.201	-0.078	2.415	-1.381	-1.419	**3.725**	1.252
Harm Avoidance	**-0.769**	-0.511	0.075	-0.294	0.156	**-3.211**	0.899	-0.309	1.067	1.095
Reward Dependence	0.126	0.164	**0.858**	0.103	-0.052	-0.414	2.080	0.001	**5.026**	2.548
Persistence	0.211	-0.178	0.107	**0.945**	0.099	-0.482	-1.801	**3.712**	1.899	0.643
Self Directedness	**0.913**	-0.052	0.154	0.174	-0.014	0.236	**2.685**	0.277	-2.361	-0.048
Cooperativeness	**0.689**	-0.200	0.539	-0.033	0.155	-0.611	**3.571**	-0.339	0.294	1.825
Self Transcendence	-0.015	-0.087	-0.022	0.084	**0.990**	1.445	-1.821	-1.237	-1.823	**3.926**
TCI Traits	FA 1	FA 2	FA 3	FA 4	FA 5					
Novelty Seeking	0.036	**0.759**	-0.171	0.066	-0.169					
Harm Avoidance	**-0.709**	-0.397	-0.346	-0.064	0.274					
Reward Dependence	0.132	0.074	0.088	**0.566**	-0.061					
Persistence	0.248	-0.215	**0.765**	0.116	0.182					
Self Directedness	**0.817**	-0.013	0.221	0.200	-0.026					
Cooperativeness	**0.672**	-0.132	-0.084	0.551	0.277					
Self Transcendence	-0.021	-0.117	0.090	-0.024	**0.528**					

Table A.18: Depressed males TCI at six months, five component solution.

self transcendence and persistence against novelty seeking. The IC solution has a first component that appears similar to PC2 with the addition of cooperativeness. The second component is essentially harm avoidance versus self directedness. The first factor is the one component (factor) solution. The second factor measures novelty seeking.

The three component solutions presented in Table A.17 show that the IC and PC methods lead to the same solution with one component measuring harm avoidance versus self directedness and cooperativeness, another component measuring novelty seeking versus persistence and the third measuring self transcendence. The factor analysis solution is quite different. The first factor is a weighted average of reward dependence and cooperativeness, the second a contrast of persistence and self directedness and the third a contrast of novelty seeking and self transcendence.

The five component PC solution (Table A.18) appears the same at baseline and six months for the depressed males apart from a change in order of the components. The IC solution also appears similar to the baseline model. The first IC is essentially harm avoidance, the second measures self directedness and cooperativeness, and the third persistence. Novelty seeking and reward dependence load highly on the fourth IC and self transcendence loads highly on the fifth. The factor model is the same as the PC model.

Comparison Across Time

At baseline, one, two and five components were rotated. The same were rotated after treatment with the addition of a four component solution. Comparison of the one component solutions (Tables A.11 and A.15) shows that the three methods, PCA, ICA and FA lead to different solutions across time. Likewise the two component solutions are different across time (Tables A.12 and A.16). The five component PC solution at baseline is the same as the PC solution after treatment, apart from an order change of the components (Tables A.13 and A.18). The IC solutions are different going from baseline to post treatment. At baseline only a four factor FA solution was rotated, after treatment a five factor solution was rotated. These solutions are different with only one component the same after treatment as at baseline. This component is essentially novelty seeking (FA 4 at baseline and FA 2 after treatment).

A.3 Comparison of the Personality for the Males and Females

At Baseline

The females had four different solutions for each of the methods, one, three, four and five component solutions; the males had three different solutions for each of the methods, one two and five component solutions. Looking at Tables A.2 and A.11, the PCA, ICA and FA solutions are different for the males compared to the females. The three solutions for the females all involve, among other variables, harm avoidance versus persistence and self directedness. The solutions for the males all involve harm avoidance versus novelty seeking and self directedness, among other variables.

Comparison of the five component solutions can also be made. These are presented in Tables A.5 and A.13. The ICA solution is the same for the males and females. The PC solution has the same last two components for both the males and females. There appears to be more similarity for the male and female solutions when a larger number of components are rotated.

After Treatment

After treatment the females had one, three and five component solutions, in comparison the males had one, two, three and five component solutions. The one component solutions, presented in Tables A.7 and A.15, have both the same PC and FA solutions across males and females. The males IC solution is also the same as the PC and FA solutions for both the males and females, and this solution contrasts harm avoidance against persistence,

	Eigenvalue	Proportion	Cumulative
1	2.580	0.512	0.512
2	0.643	0.128	0.640
3	0.491	0.098	0.737
4	0.377	0.075	0.812
5	0.310	0.062	0.873
6	0.246	0.049	0.922
7	0.155	0.031	0.953
8	0.125	0.025	0.978
9	0.113	0.023	1.000

Table A.19: Eigenvalues and variances for the symptoms of the depressed males and females at baseline.

SCL Symptoms	PC 1	IC 1	FA 1
Somatisation	**0.632**	**-0.163**	**0.640**
Obsessive Compulsive	**0.668**	**-0.201**	**0.632**
Interpersonal Sensitivity	**0.816**	**-0.270**	**0.728**
Depression	**0.756**	**-0.210**	**0.740**
Anxiety	**0.719**	**-0.193**	**0.732**
Anger Hostility	**0.521**	**-0.164**	**0.422**
Phobic Anxiety	**0.721**	**-0.215**	**0.661**
Paranoid Ideation	**0.767**	**-0.249**	**0.671**
Psychotocism	**0.798**	**-0.184**	**0.819**

Table A.20: Depressed males and females SCL at baseline, one component solution.

self directedness and cooperativeness. The females IC solution is different to the rest and contrasts harm avoidance against persistence, self directedness and self transcendence.

The three component solutions are different across gender, these solutions are presented in Tables A.8 and A.17. The five components (from Tables A.9 and A.18) are the same for the male and female PC solutions. However, the IC and FA solutions lead to different structures for the males compared to the females.

A.4 The Depressed Males and Females Symptoms

Baseline Results

As the depressed males and females have a common component structure at baseline, the data has been combined and models calculated accordingly with the males and females combined. The combined eigenvalues are shown in Table A.19. The first eigenvalue accounts for 51.2% of the variance and six components are needed to retain 90% of the variance.

The one factor solution is shown in Table A.20, and is essentially an average across all the traits with interpersonal sensitivity loading the highest for both the PC and IC solutions. The first PC of the two component solution (Table A.21) is also an average of

SCL Symptoms	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
Somatisation	**0.725**	0.144	-0.031	**0.428**	**0.722**	0.192
Obsessive Compulsive	**0.752**	0.168	-0.046	**0.502**	**0.536**	0.335
Interpersonal Sensitivity	0.408	**0.764**	**-0.376**	-0.279	0.186	**0.938**
Depression	**0.717**	0.335	-0.117	**0.317**	**0.537**	0.513
Anxiety	**0.853**	0.133	-0.022	**0.548**	**0.850**	0.236
Anger Hostility	0.057	**0.709**	-0.346	**-0.529**	0.220	**0.342**
Phobic Anxiety	**0.563**	0.454	-0.188	0.119	0.420	**0.532**
Paranoid Ideation	0.270	**0.840**	-0.414	**-0.463**	0.254	**0.702**
Psychotocism	**0.672**	0.448	-0.139	0.166	**0.603**	0.512

Table A.21: Depressed males and females SCL at baseline, two component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	IC 1	IC 2	IC 3	IC 4
Somatisation	0.099	**0.836**	0.203	0.150	**-0.810**	-0.404	0.001	0.102
Obsessive Compulsive	0.126	0.245	**0.863**	0.134	0.540	0.172	-0.041	**0.956**
Interpersonal Sensitivity	**0.882**	0.133	0.312	0.082	0.255	**0.658**	0.347	-0.293
Depression	0.407	0.278	**0.699**	0.024	0.290	0.390	0.030	**0.498**
Anxiety	0.143	**0.756**	0.453	0.066	**-0.556**	-0.163	-0.049	0.349
Anger Hostility	0.211	0.131	0.110	**0.943**	0.394	**-1.090**	0.547	0.050
Phobic Anxiety	0.574	**0.626**	0.060	-0.001	**-0.791**	0.107	0.147	-0.396
Paranoid Ideation	**0.800**	0.164	0.146	0.331	0.148	0.150	**0.467**	-0.424
Psychotocism	0.448	**0.508**	0.410	0.169	-0.172	0.017	0.109	0.112
SCL Symptoms	FA 1	FA 2	FA 3	FA 4				
Somatisation	**0.772**	0.119	0.102	0.219				
Obsessive Compulsive	0.407	**0.429**	0.180	0.188				
Interpersonal Sensitivity	0.126	0.301	**0.852**	0.409				
Depression	0.289	**0.830**	0.260	0.204				
Anxiety	**0.743**	0.400	0.128	0.142				
Anger Hostility	0.158	0.112	0.107	**0.480**				
Phobic Anxiety	0.431	0.185	**0.453**	0.226				
Paranoid Ideation	0.193	0.154	0.398	**0.721**				
Psychotocism	**0.525**	0.336	0.291	0.413				

Table A.22: Depressed males and females SCL at baseline, four component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
Somatisation	**0.890**	0.164	0.032	0.147	0.132	0.082
Obsessive Compulsive	0.261	0.148	0.235	0.126	**0.910**	0.099
Interpersonal Sensitivity	0.036	**0.722**	0.435	0.408	0.142	0.099
Depression	0.279	0.235	**0.847**	0.130	0.246	0.090
Anxiety	**0.764**	0.073	0.417	0.197	0.198	0.074
Anger Hostility	0.111	0.206	0.082	0.088	0.087	**0.960**
Phobic Anxiety	0.292	0.238	0.138	**0.890**	0.124	0.101
Paranoid Ideation	0.243	**0.902**	0.074	0.101	0.093	0.220
Psychotocism	**0.538**	0.433	0.334	0.181	0.225	0.133
SCL Symptoms	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
Somatisation	-0.333	0.284	-0.114	**0.875**	-0.136	0.548
Obsessive Compulsive	0.551	-0.004	-0.274	0.440	**1.332**	-0.644
Interpersonal Sensitivity	-0.294	0.007	-0.278	**-0.713**	-0.043	-0.324
Depression	-0.008	-0.363	0.209	**-1.152**	0.177	0.836
Anxiety	-0.119	0.184	0.119	0.055	-0.037	**0.842**
Anger Hostility	**1.129**	-0.376	-0.350	0.001	-0.566	0.262
Phobic Anxiety	0.426	**1.350**	0.021	-0.176	-0.371	-0.485
Paranoid Ideation	**-0.742**	-0.435	-0.704	0.546	-0.106	-0.337
Psychotocism	-0.237	-0.042	-0.145	0.121	0.035	0.290

Table A.23: Depressed males and females SCL at baseline, six component solution.

six symptoms. Interpersonal sensitivity, anger hostility and paranoid ideation load high on the second PC. The first IC measures interpersonal sensitivity. The second IC contrasts somatisation, obsessive compulsive, depression and anxiety against anger hostility and paranoid ideation. The first factor (FA 1) is a weighted average of somatisation, obsessive compulsive, depression, anxiety and psychotocism. The second factor (FA 2) is a weighted average of interpersonal sensitivity, anger hostility, phobic anxiety and paranoid ideation.

The first PC in the four component solution (Table A.22) has high loadings for interpersonal sensitivity and paranoid ideation. The second principal component is comprised of somatisation, anxiety, phobic anxiety, and psychotocism. Obsessive compulsive and depression symptoms are measured in the third component, whilst anger hostility is essentially the fourth. For the IC's the first component is made up of somatisation, anxiety and phobic anxiety; the second IC contrasts anger hostility with interpersonal sensitivity; the third is essentially paranoid ideation and the fourth obsessive compulsive and interpersonal sensitivity. The first factor combines somatisation, anxiety and psychotocism; the second obsessive compulsive and depression; the third interpersonal sensitivity and phobic anxiety and the fourth measures anger hostility and paranoid ideation.

The first PC in the six component solution is a mixture of somatisation, anxiety and psychotocism; the second combines paranoid ideation and interpersonal sensitivity. The third PC is an indicator of depression; the fourth phobic anxiety and the fifth obsessive compulsive. Anger hostility is measured by the sixth principal component. IC1 contrasts anger hostility against paranoid ideation. IC2 measures phobic anxiety. No variables have their highest loading on IC3 making it a redundant component. IC4 contrasts depression and interpersonal sensitivity against somatisation. IC5 measures obsessive compulsive and IC6 measures anxiety. The SAS (SAS(R) Proprietary Software Release (8.1)) criteria suggested that six was too many factors for the factor analysis to be conducted.

A.5 The Depressed Females Symptoms

The Flury test from Chapter 3 showed that there were no common components across males and females symptoms after treatment. Thus males and females are treated separately for their post treatment symptoms.

Post Treatment Results

At six months the first component of the depressed female's symptoms accounts for 71% of the variance and only four components are required to account for 90% of the variance (Table A.24). The one component solution, shown in Table A.25, for the depressed females at six months is an average of all symptoms with depression as the highest loading

	Eigenvalue	Proportion	Cumulative
1	1.343	0.710	0.710
2	0.173	0.092	0.801
3	0.117	0.062	0.863
4	0.082	0.043	0.906
5	0.063	0.034	0.940
6	0.045	0.024	0.964
7	0.030	0.016	0.980
8	0.024	0.013	0.992
9	0.015	0.008	1.000

Table A.24: Eigenvalues and variances for the symptoms of the depressed females at six months.

SCL Symptoms	PC 1	IC 1	FA 1
Somatisation	**0.674**	**0.173**	**0.665**
Obsessive Compulsive	**0.900**	**0.389**	**0.890**
Interpersonal Sensitivity	**0.819**	**0.296**	**0.782**
Depression	**0.959**	**0.494**	**0.938**
Anxiety	**0.905**	**0.300**	**0.905**
Anger Hostility	**0.736**	**0.258**	**0.690**
Phobic Anxiety	**0.587**	**0.117**	**0.558**
Paranoid Ideation	**0.676**	**0.222**	**0.637**
Psychotocism	**0.814**	**0.154**	**0.810**

Table A.25: Depressed females SCL at six months, one component solution.

symptom, for all three methods.

In the two component solution (Table A.26) PC 1 combines somatisation, obsessive compulsive, depression, anxiety and anger hostility symptoms. PC 2 measures interpersonal sensitivity, phobic anxiety, paranoid ideation and psychotocism. The first IC combines somatisation, obsessive compulsive, anxiety and anger hostility. The second IC is essentially interpersonal sensitivity, depression and paranoid ideation. The factor analysis solution is the same as the PC solution.

The three component solution for the depressed females at six months is shown in Table A.27. The first PC combines somatisation, obsessive compulsive symptoms, depression and anxiety. The second PC is a combination of interpersonal sensitivity, paranoid

SCL Symptoms	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
Somatisation	**0.735**	0.173	**-0.573**	-0.409	**0.678**	0.223
Obsessive Compulsive	**0.884**	0.348	**-0.954**	-0.525	**0.823**	0.407
Interpersonal Sensitivity	0.390	**0.809**	0.440	**0.978**	0.378	**0.787**
Depression	**0.693**	0.666	-0.215	**0.511**	**0.676**	0.647
Anxiety	**0.873**	0.368	**-0.695**	-0.357	**0.894**	0.369
Anger Hostility	**0.674**	0.343	**-0.499**	-0.191	**0.575**	0.374
Phobic Anxiety	0.349	**0.497**	0.066	0.260	0.306	**0.507**
Paranoid Ideation	0.132	**0.893**	0.813	**1.304**	0.208	**0.759**
Psychotocism	0.461	**0.716**	0.119	0.379	0.462	**0.733**

Table A.26: Depressed females SCL at six months, two component solution.

SCL Symptoms	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
Somatisation	**0.741**	0.153	0.170	**-0.782**	-0.137	0.369	**0.672**	0.185	0.166
Obsessive Compulsive	**0.882**	0.318	0.247	**-1.208**	-0.300	0.757	**0.822**	0.384	0.150
Interpersonal Sensitivity	0.346	**0.768**	0.311	**1.010**	-0.178	0.313	0.368	**0.693**	0.396
Depression	**0.635**	0.612	0.383	0.246	0.031	**0.518**	**0.676**	0.611	0.210
Anxiety	**0.825**	0.318	0.349	**-0.743**	0.114	0.372	**0.887**	0.339	0.173
Anger Hostility	0.353	0.176	**0.906**	0.337	**2.386**	-1.046	**0.568**	0.265	0.346
Phobic Anxiety	0.183	0.407	**0.522**	0.457	**0.587**	-0.242	0.251	0.249	**0.938**
Paranoid Ideation	0.133	**0.878**	0.172	**1.403**	-0.545	0.380	0.204	**0.766**	0.146
Psychotocism	0.492	**0.707**	0.148	0.240	-0.390	0.359	0.456	**0.739**	0.163

Table A.27: Depressed females SCL at six months, three component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	IC 1	IC 2	IC 3	IC 4
Somatisation	**0.744**	0.112	0.121	0.144	**0.821**	-0.184	0.024	0.255
Obsessive Compulsive	**0.886**	0.213	0.287	0.175	**1.205**	-0.344	-0.394	0.749
Interpersonal Sensitivity	0.349	0.452	**0.800**	0.085	-1.339	0.009	**-1.949**	1.206
Depression	**0.652**	0.557	0.321	0.314	-0.014	-0.031	**1.086**	0.183
Anxiety	**0.833**	0.260	0.234	0.299	**0.820**	0.058	0.185	0.210
Anger Hostility	0.358	0.162	0.291	**0.863**	-0.271	**2.389**	0.319	-1.119
Phobic Anxiety	0.177	0.126	**0.712**	0.326	-0.676	0.700	**-1.267**	0.299
Paranoid Ideation	0.164	**0.917**	0.194	0.141	-1.061	-0.605	**2.003**	-0.134
Psychotocism	0.506	**0.578**	0.416	0.038	-0.222	-0.381	0.035	0.396
SCL Symptoms	FA 1	FA 2	FA 3	FA 4				
Somatisation	**0.686**	0.177	0.173	0.000				
Obsessive Compulsive	**0.842**	0.380	0.156	0.018				
Interpersonal Sensitivity	0.368	**0.674**	0.402	0.058				
Depression	**0.643**	0.617	0.208	0.320				
Anxiety	**0.857**	0.354	0.181	0.121				
Anger Hostility	**0.536**	0.243	0.350	0.318				
Phobic Anxiety	0.229	0.248	**0.940**	0.050				
Paranoid Ideation	0.183	**0.747**	0.151	0.158				
Psychotocism	0.449	**0.799**	0.161	-0.126				

Table A.28: Depressed females SCL at six months, four component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Somatisation	0.332	0.156	0.160	**0.841**	0.164	0.382	-0.167	0.804	0.916	**2.793**
Obsessive Compulsive	**0.779**	0.250	0.125	0.468	0.198	-0.559	0.216	-0.909	-0.441	**0.953**
Interpersonal Sensitivity	0.445	**0.781**	0.386	0.090	0.086	**-1.681**	0.045	1.572	-1.322	-0.051
Depression	**0.761**	0.273	0.440	0.132	0.326	0.732	0.221	-1.247	-0.872	**-1.555**
Anxiety	**0.752**	0.198	0.173	0.411	0.321	0.028	-0.113	**-0.810**	-0.100	0.326
Anger Hostility	0.302	0.287	0.140	0.191	**0.870**	0.407	**-2.343**	0.369	1.057	-0.600
Phobic Anxiety	0.129	**0.724**	0.129	0.178	0.325	-1.000	-0.730	**1.253**	-0.129	0.376
Paranoid Ideation	0.204	0.216	**0.922**	0.141	0.135	**2.366**	0.555	1.229	0.415	0.594
Psychotocism	**0.566**	0.392	0.505	0.184	0.045	0.065	0.386	0.159	-0.413	0.064
SCL Symptoms	FA 1	FA 2	FA 3	FA 4	FA 5					
Somatisation	0.251	0.133	0.170	**0.750**	0.193					
Obsessive Compulsive	**0.645**	0.165	0.249	0.534	0.282					
Interpersonal Sensitivity	0.407	0.353	**0.781**	0.161	0.152					
Depression	**0.604**	0.389	0.336	0.325	0.384					
Anxiety	**0.644**	0.177	0.198	0.522	0.390					
Anger Hostility	0.258	0.153	0.265	0.250	**0.726**					
Phobic Anxiety	0.096	0.146	**0.579**	0.198	0.379					
Paranoid Ideation	0.191	**0.930**	0.236	0.131	0.159					
Psychotocism	**0.568**	0.475	0.395	0.239	0.093					

Table A.29: Depressed females SCL at six months, five component solution.

ideation and psychotocism. Anger hostility and phobic anxiety are measured in the third principal component. The first IC contrasts somatisation, obsessive compulsive symptoms and anxiety against interpersonal sensitivity and paranoid ideation. The second IC measures anger hostility and phobic anxiety, the third depression. The first factor (FA 1) is a weighted average of somatisation, obsessive compulsive, depression, anxiety and anger hostility. FA 2 is an average of interpersonal sensitivity, paranoid ideation and psychotocism. FA 3 is essentially phobic anxiety.

For the four component solution (Table A.28), the first PC is the same as the first PC in the three component solution. PC 2 measures paranoid ideation and psychotocism. PC 3 combines interpersonal sensitivity and phobic anxiety and the fourth PC anger hostility. Table A.28 also shows the independent component loadings. The first IC combines somatisation, obsessive compulsive and anxiety, the second is essentially anger hostility. IC 3 contrasts paranoid ideation and depression against interpersonal sensitivity and phobic anxiety. No variables have their highest loading on the fourth IC so it is redundant. The four factor solution is the same as the three factor solution, no variables have their highest loading on the fourth factor so it reduces to the three factor solution.

Finally the five component solution at six months, shown in Table A.29 has a first PC that is a weighted average of obsessive compulsive, depression, anxiety and psychotocism. The second PC is a combination of interpersonal sensitivity and phobic anxiety. Paranoid ideation loads highly on PC 3. PC 4 is essentially somatisation, whilst PC 5 is anger hostility. The first IC contrasts paranoid ideation against interpersonal sensitivity. The second IC is essentially anger hostility. IC3 contrasts anxiety against phobic anxiety. IC4 is redundant with no variables loading highest on it. IC5 is a contrast of somatisation and obsessive compulsive symptoms against depression. The five component FA model is the same as the five component PC model.

It is interesting to note that all the PC and FA symptom models have positive loadings suggesting that the symptoms are measured in a "similar direction", however the IC models in some cases have contrasting loadings. This suggests that to obtain independence rather than just decorrelated constructs the symptoms are not all measured in a similar direction.

Comparison Across Time

At baseline, one, two, four and six components were retained, whereas after treatment one through five components were retained. The one component solutions are presented in Tables A.20 and A.25. All three methods at both time points lead to a solution that is a weighted average of all the symptom variables. At baseline interpersonal sensitivity loads the highest for the PC and IC methods. The FA solution has a highest loading for the symptom psychotocism. After treatment the highest loading symptom, across all

	Eigenvalue	Proportion	Cumulative
1	2.759	0.733	0.733
2	0.299	0.080	0.812
3	0.214	0.057	0.869
4	0.182	0.048	0.918
5	0.115	0.031	0.948
6	0.085	0.023	0.971
7	0.053	0.014	0.985
8	0.037	0.010	0.994
9	0.021	0.006	1.000

Table A.30: Eigenvalues and variances for the symptoms of the depressed males at six months.

SCL Symptoms	PC 1	IC 1	FA 1
Somatisation	**0.767**	**0.145**	**0.785**
Obsessive Compulsive	**0.874**	**0.227**	**0.827**
Interpersonal Sensitivity	**0.929**	**0.281**	**0.894**
Depression	**0.926**	**0.265**	**0.906**
Anxiety	**0.901**	**0.224**	**0.897**
Anger Hostility	**0.678**	**0.158**	**0.632**
Phobic Anxiety	**0.719**	**0.149**	**0.685**
Paranoid Ideation	**0.837**	**0.175**	**0.834**
Psychotocism	**0.890**	**0.145**	**0.912**

Table A.31: Depressed males symptoms at six months, one component solution.

three methods, is the symptom of depression.

Comparison of the two component solutions, presented in Tables A.21 and A.26, shows that the solutions are different across time. Likewise the four component solutions are different across time (Tables A.22 and A.26).

A.6 The Depressed Males Symptoms

Post Treatment Results

Table A.30 presents the information about the eigenvalues and percentage variance accounted for, for the depressed males after treatment. The first eigenvalue accounts for 73% of the variance and only four components need to be retained to keep 90% of the variance. The one component solution, presented in Table A.31, is a weighted average of all symptoms. The three methods all give this weighted average structure.

The loadings for the three component solutions are presented in Table A.32. The first PC is an average of somatisation, obsessive compulsive, depression, anxiety, paranoid ideation and psychotocism. Somatisation is the highest loading symptom on this factor. PC 2 is a combination of phobic anxiety and interpersonal sensitivity. Anger hostility is measured by the third PC. The first IC measures a contrast of somatisation

SCL Symptoms	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
Somatisation	**0.896**	0.104	0.208	**0.854**	-0.752	0.364	**0.717**	0.452	0.095
Obsessive Compulsive	**0.657**	0.378	0.468	0.328	0.116	**0.333**	0.376	**0.765**	0.299
Interpersonal Sensitivity	0.517	**0.752**	0.309	**-0.816**	0.038	0.064	0.440	**0.578**	0.577
Depression	**0.798**	0.452	0.261	0.359	**-0.719**	0.350	0.529	**0.683**	0.346
Anxiety	**0.634**	0.606	0.254	-0.230	-0.333	0.156	**0.604**	0.414	0.537
Anger Hostility	0.268	0.169	**0.927**	0.282	**1.741**	0.308	0.408	**0.480**	0.160
Phobic Anxiety	0.167	**0.931**	0.135	**-1.307**	0.193	-0.207	0.195	0.215	**0.957**
Paranoid Ideation	**0.550**	0.420	0.497	0.072	**0.314**	0.214	**0.679**	0.365	0.373
Psychotocism	**0.726**	0.434	0.319	0.171	-0.238	0.190	**0.832**	0.371	0.341

Table A.32: Depressed males symptoms at six months, three component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	IC 1	IC 2	IC 3	IC 4
Somatisation	**0.883**	0.078	0.323	0.178	-0.790	-0.473	**-0.848**	-0.624
Obsessive Compulsive	0.339	0.272	**0.771**	0.371	-0.438	-0.312	0.453	**1.304**
Interpersonal Sensitivity	0.344	**0.687**	0.540	0.247	**0.767**	-0.167	0.295	0.514
Depression	0.564	0.365	**0.671**	0.180	-0.423	**-0.893**	-0.048	0.526
Anxiety	**0.655**	0.592	0.261	0.234	0.316	-0.032	-0.684	**-0.782**
Anger Hostility	0.239	0.150	0.254	**0.906**	-0.219	**1.721**	-0.153	0.467
Phobic Anxiety	0.162	**0.919**	0.187	0.120	**1.323**	0.218	0.144	-0.214
Paranoid Ideation	**0.599**	0.416	0.200	0.487	0.021	0.570	**-0.624**	-0.523
Psychotocism	**0.733**	0.414	0.297	0.295	-0.113	-0.034	**-0.539**	-0.473
SCL Symptoms	FA 1	FA 2	FA 3	FA 4				
Somatisation	**0.765**	0.093	0.340	0.357				
Obsessive Compulsive	0.324	0.295	**0.684**	0.351				
Interpersonal Sensitivity	0.299	**0.612**	0.571	0.323				
Depression	0.523	0.365	**0.681**	0.219				
Anxiety	**0.625**	0.591	0.312	0.249				
Anger Hostility	0.266	0.172	0.398	**0.437**				
Phobic Anxiety	0.125	**0.879**	0.214	0.211				
Paranoid Ideation	0.365	0.329	0.272	**0.828**				
Psychotocism	**0.612**	0.368	0.365	0.478				

Table A.33: Depressed males symptoms at six months, four component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
Somatisation	**0.865**	0.081	0.287	0.159	0.244	**0.882**	0.732	-0.367	0.545	-0.474
Obsessive Compulsive	0.330	0.262	**0.779**	0.308	0.248	-0.553	0.407	0.526	**-1.189**	-0.935
Interpersonal Sensitivity	0.330	**0.684**	0.530	0.210	0.210	-0.328	**-0.770**	0.335	-0.417	-0.312
Depression	0.612	0.399	**0.613**	0.223	-0.007	-0.798	0.545	0.430	**1.025**	-0.500
Anxiety	**0.664**	0.618	0.198	0.276	0.080	0.085	-0.238	-0.539	**1.459**	0.450
Anger Hostility	0.223	0.160	0.242	**0.914**	0.164	-1.155	0.332	**-1.219**	-0.099	1.155
Phobic Anxiety	0.135	**0.913**	0.177	0.097	0.163	-0.032	**-1.317**	0.004	0.041	0.359
Paranoid Ideation	0.470	0.353	0.251	0.317	**0.680**	**1.526**	-0.251	-0.737	-1.435	-0.366
Psychotocism	**0.686**	0.402	0.280	0.242	0.348	**0.678**	0.051	-0.376	0.084	-0.168
SCL Symptoms	FA 1	FA 2	FA 3	FA 4	FA 5					
Somatisation	**0.769**	0.105	0.338	0.328	0.0417					
Obsessive Compulsive	0.339	0.318	**0.763**	0.272	0.007					
Interpersonal Sensitivity	0.355	**0.678**	0.486	0.267	-0.133					
Depression	**0.599**	0.426	0.576	0.153	-0.137					
Anxiety	0.640	**0.627**	0.273	0.215	0.277					
Anger Hostility	0.261	0.193	**0.477**	0.390	0.169					
Phobic Anxiety	0.130	**0.844**	0.196	0.211	0.033					
Paranoid Ideation	0.383	0.342	0.308	**0.801**	-0.009					
Psychotocism	**0.659**	0.406	0.295	0.445	-0.060					

Table A.34: Depressed males symptoms at six months, five component solution.

SCL Symptoms	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
Somatisation	**0.868**	0.085	0.159	0.260	0.257	0.086
Obsessive Compulsive	0.356	0.291	0.301	**0.792**	0.210	0.155
Interpersonal Sensitivity	0.306	**0.603**	0.225	0.355	0.303	0.502
Depression	**0.604**	0.341	0.237	0.442	0.069	0.484
Anxiety	**0.666**	0.633	0.274	0.173	0.100	0.107
Anger Hostility	0.220	0.145	**0.916**	0.211	0.184	0.102
Phobic Anxiety	0.134	**0.924**	0.092	0.173	0.174	0.102
Paranoid Ideation	0.452	0.322	0.317	0.229	**0.710**	0.097
Psychotocism	**0.666**	0.354	0.250	0.179	0.415	0.262
SCL Symptoms	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
Somatisation	**0.834**	0.602	0.098	-0.739	0.380	0.601
Obsessive Compulsive	0.612	**1.591**	0.472	1.082	**0.818**	-1.420
Interpersonal Sensitivity	-1.002	**-1.556**	-0.515	-0.411	0.312	-0.566
Depression	0.382	**-1.502**	0.483	0.169	**0.729**	0.539
Anxiety	-0.066	1.030	0.770	0.411	-0.268	**1.561**
Anger Hostility	0.203	-0.412	**-1.144**	1.294	-0.801	0.707
Phobic Anxiety	**-1.161**	1.151	0.501	0.495	-0.360	0.081
Paranoid Ideation	-0.222	0.721	-1.416	**-1.432**	-0.025	-0.811
Psychotocism	0.018	-0.175	-0.451	**-0.828**	0.082	0.266

Table A.35: Depressed males symptoms at six months, six component solution

against phobic anxiety and interpersonal sensitivity. The second IC contrasts anger hostility and paranoid ideation against somatisation. The third IC is a measure of obsessive compulsive symptoms. The first factor measures somatisation, anxiety, paranoid ideation and psychotocism. The second measures obsessive compulsive, interpersonal sensitivity, depression and anger hostility. Phobic anxiety is the sole indicator of the third factor.

The four component solution is shown in Table A.33. The first principal component measures somatisation, psychotocism, paranoid ideation and anxiety. The second PC combines phobic anxiety and interpersonal sensitivity. Obsessive compulsive and depression are important in the third PC. Finally, the fourth PC measures anger hostility. The first IC measures phobic anxiety and interpersonal sensitivity. The second IC contrasts anger hostility against depression. Somatisation, paranoid ideation and psychotocism are measured in the third IC whilst the fourth IC measures obsessive compulsive versus anxiety. The first factor measures somatisation, anxiety and psychotocism; the second interpersonal sensitivity and phobic anxiety; and the third obsessive compulsive and depression. Anger hostility and paranoid ideation load highest on the fourth factor.

Looking at the five component solutions presented in Table A.34, the first PC measures somatisation, anxiety and psychotocism. The second principal component is the same across the four, five and six component solutions. The third and fourth PCs are the same as the third and fourth PCs of the four factor solution. PC5 measures paranoid ideation. For the independent components, retaining five components results in a first component measuring somatisation, paranoid ideation and psychotocism, a second component measuring phobic anxiety and a third component measuring anger hostility.

A contrast is measured in the fourth IC with obsessive compulsive symptoms versus depression and anxiety. The fifth component is redundant. The five factor solution also has a redundant fifth factor.

The six component solutions for the PCs and ICs are presented in Table A.35. There were too many components retained for factor analysis. The sixth component is redundant in the PC solution.

Comparison Across Time

At baseline the combined gender model has one, two, four and six component solutions. After treatment the males had one, three, four, five and six component solutions. The one component solutions are presented in Tables A.20 and A.31. At both time points the one component solutions are weighted averages of all the symptoms. Interpersonal sensitivity was the highest loading symptom for the IC and PC solutions; whereas psychotocism was the highest loading symptom for the FA solutions. After treatment the highest loading symptoms remain the same.

Comparison of the four component solutions, presented in Tables A.22 and A.33, shows that the FA solution is the same at both time points. The PC solution has two components the same across time and two components that differ. The IC solution has one common component across time with three that differ. This is different to the female comparison across time where all the components differed.

The last comparison is between the six component solutions, presented in Tables A.23 and A.35. Six components were too many for a factor solution to be obtained. The PC solution has two common components across time. The post treatment solution also has one redundant component. The IC solution has one component that is similar across time and the baseline IC solution has one redundant component.

A.7 Comparison of the Symptoms for the Males and Females

After Treatment

The component solutions for the depressed females had one to five components retained, in comparison to the component solutions for the depressed males, which retained one, three, four, five and six components. The one component solutions (Tables A.25 and A.31), for both the males and females, represent weighted averages of all the symptoms. The highest loading symptom for all three methods for the females is that of depression. For the males, interpersonal sensitivity is the highest loading symptom for the PC and

	Eigenvalue	Proportion	Cumulative
1	2.479	0.476	0.476
2	0.712	0.137	0.613
3	0.513	0.099	0.712
4	0.389	0.075	0.787
5	0.325	0.062	0.849
6	0.210	0.040	0.889
7	0.178	0.034	0.924
8	0.120	0.023	0.947
9	0.093	0.018	0.965
10	0.059	0.011	0.976
11	0.045	0.009	0.985
12	0.027	0.005	0.990
13	0.023	0.004	0.994
14	0.016	0.003	0.997
15	0.008	0.002	0.999
16	0.005	0.001	1.000

Table A.36: Eigenvalues and variances for the personality and symptoms of the depressed females at baseline.

IC methods, the FA method has psychotocism as the highest loading symptom. So whilst the one component solutions are similar across males and females after treatment the importance of the symptoms within the component is different across gender.

The three component solutions are presented in Tables A.27 and A.32. The solutions are different for the males and females. Likewise for the four component solutions (Tables A.28 and A.33) the components are different across males and females. The five component solutions, shown in Tables A.29 and A.34, have one similar component across gender. The similar component is found in the PC solutions.

A.8 Depressed Females Personality and Symptoms

Component models were developed on all 16 variables to allow investigation of the covariance structure across the symptom and personality traits combined.

Baseline Results

For the baseline females, seven components are needed to retain at least 90% of the variance (Table A.36). Table A.37 presents the two component solutions. The PC solution has a first component purely made up of symptom variables. The second component has self directedness contrasting against the symptom variables. The IC solution has both components made up purely of symptom variables. The factor analysis model has a first component that is a weighted average of most symptoms and a second component that contrasts personality against symptoms.

Variables	PC1	PC2	IC 1	IC 2	FA 1	FA 2
NS TCI	0.108	0.132	0.013	-0.003	0.126	0.083
HA TCI	0.014	0.302	0.030	-0.052	-0.014	**0.473**
RD TCI	0.087	-0.042	-0.003	0.022	0.118	-0.087
P TCI	0.035	-0.259	-0.038	0.079	0.061	**-0.370**
S TCI	-0.123	**-0.519**	-0.057	0.076	-0.130	**-0.668**
C TCI	0.025	-0.370	-0.025	0.049	0.015	**-0.392**
ST TCI	0.377	0.127	0.014	0.041	**0.448**	-0.058
S SCL	**0.788**	0.097	0.067	**0.476**	**0.773**	-0.017
OC SCL	**0.798**	0.072	0.065	**0.559**	**0.609**	0.138
IS SCL	0.321	**0.834**	0.390	**-0.408**	0.411	**0.787**
D SCL	**0.744**	0.396	0.184	0.262	**0.720**	0.359
A SCL	**0.857**	0.084	0.063	**0.509**	**0.872**	-0.48
AH SCL	0.173	**0.623**	0.274	**-0.343**	0.311	**0.364**
PA SCL	0.415	**0.565**	**0.247**	-0.098	**0.458**	0.407
PI SCL	0.253	**0.807**	0.328	**-0.389**	0.425	**0.584**
P SCL	**0.677**	0.414	0.145	0.157	**0.718**	0.349

Table A.37: Depressed females TCI and SCL at baseline, two component solution.

Variables	PC1	PC2	PC3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
NS TCI	0.110	0.008	0.272	0.064	0.000	0.047	0.119	0.269	**-0.371**
HA TCI	-0.022	0.354	-0.012	-0.053	0.067	-0.031	0.052	0.208	**0.977**
RD TCI	0.092	-0.042	0.003	0.002	-0.021	0.011	0.122	-0.218	0.082
P TCI	0.055	-0.210	-0.147	-0.024	-0.085	-0.003	0.015	**-0.355**	-0.154
S TCI	-0.081	**-0.446**	-0.284	-0.016	-0.092	-0.020	-0.211	**-0.606**	-0.422
C TCI	0.041	-0.190	**-0.412**	-0.065	-0.047	-0.032	-0.011	**-0.580**	0.095
ST TCI	0.383	-0.011	0.343	0.070	-0.039	0.078	**0.430**	-0.021	-0.211
S SCL	**0.773**	0.189	-0.007	-0.250	-0.362	0.208	**0.763**	-0.118	0.022
OC SCL	**0.797**	0.064	0.167	-0.003	**-0.469**	0.396	**0.616**	0.098	-0.036
IS SCL	0.235	**0.890**	0.167	-0.409	**0.599**	-0.091	0.517	**0.591**	0.247
D SCL	**0.710**	0.396	0.227	-0.101	-0.135	0.279	**0.761**	0.233	0.082
A SCL	**0.846**	0.156	0.038	-0.191	**-0.401**	0.257	**0.859**	-0.161	0.011
AH SCL	0.162	0.200	**0.941**	**1.022**	0.303	0.643	0.340	**0.443**	-0.029
PA SCL	0.346	**0.706**	-0.037	**-0.525**	0.271	-0.113	**0.511**	0.274	0.142
PI SCL	0.181	**0.755**	0.341	-0.070	**0.510**	0.052	0.497	**0.555**	-0.057
P SCL	**0.639**	0.429	0.193	-0.112	-0.058	0.173	**0.757**	0.284	0.007

Table A.38: Depressed females TCI and SCL at baseline, three component solution.

Variables	PC1	PC2	PC3	PC4	IC 1	IC 2	IC 3	IC 4
NS TCI	0.019	0.030	0.094	0.276	0.056	-0.034	0.011	0.043
HA TCI	0.371	0.039	-0.034	-0.037	-0.023	0.096	0.041	-0.028
RD TCI	-0.033	0.020	0.094	0.006	0.003	-0.002	-0.020	0.014
P TCI	-0.333	0.185	-0.051	-0.090	-0.103	-0.205	-0.025	-0.021
S TCI	**-0.520**	-0.007	-0.093	-0.242	-0.064	-0.125	-0.059	-0.025
C TCI	-0.231	0.052	0.034	-0.392	-0.071	-0.004	-0.047	-0.027
ST TCI	-0.046	0.225	0.288	0.378	0.045	-0.088	-0.010	0.072
S SCL	-0.060	**0.813**	0.409	0.114	-0.550	**-0.765**	-0.103	0.145
OC SCL	0.237	0.092	**0.873**	0.133	0.263	0.580	**-0.621**	0.546
IS SCL	**0.898**	0.289	0.123	0.134	-0.169	**0.755**	0.411	-0.074
D SCL	0.479	0.272	**0.668**	0.210	0.086	**0.450**	-0.241	0.362
A SCL	0.012	**0.665**	0.584	0.123	-0.351	**-0.433**	-0.244	0.248
AH SCL	0.184	0.129	0.059	**0.960**	**0.854**	-0.630	0.501	0.527
PA SCL	0.492	**0.672**	0.019	0.028	**-0.666**	-0.213	0.371	-0.192
PI SCL	**0.660**	0.404	-0.028	0.356	-0.088	0.023	**0.532**	-0.018
P SCL	0.369	**0.486**	0.455	0.226	-0.122	**-0.025**	-0.026	0.175

Variables	FA 1	FA 2	FA 3	FA 4
NS TCI	0.157	-0.259	0.041	**0.699**
HA TCI	-0.001	**0.889**	-0.031	-0.095
RD TCI	0.138	0.040	-0.191	-0.099
P TCI	0.020	-0.269	-0.034	**-0.499**
S TCI	-0.156	**-0.638**	-0.263	-0.317
C TCI	0.019	-0.110	-0.348	**-0.421**
ST TCI	**0.456**	-0.203	0.026	0.111
S SCL	**0.758**	0.003	0.028	-0.102
OC SCL	**0.644**	0.064	-0.023	0.195
IS SCL	0.436	0.491	**0.505**	0.157
D SCL	**0.747**	0.241	0.144	0.099
A SCL	**0.849**	-0.007	0.036	-0.137
AH SCL	0.314	0.104	0.333	**0.347**
PA SCL	**0.466**	0.247	0.268	0.054
PI SCL	0.385	0.129	**0.914**	0.001
P SCL	**0.733**	0.159	0.241	0.180

Table A.39: Depressed females TCI and SCL at baseline, four component solution.

The three component solutions are presented in Table A.38. The pattern across the three methods is again similar. ICA has no contributions from the personality variables, PCA has few and FA has the most. Table A.39 presents the four component solutions. The PCA solution models firstly self directedness versus interpersonal sensitivity and paranoid ideation, then there are two components with weighted averages of symptoms and a final component measuring anger hostility. The ICA solution, like the three component solution, has a redundant component and measures only symptom variables. The factor analysis model has three components mixing personality and symptoms, and one symptom specific component.

Table A.40 presents the seven component solutions. The first principal component is an average of somatisation, anxiety and psychotocism; the second component is a mixture of personality and symptoms and the last five components are single symptom indicator components. The ICA solution has two redundant components, and still only measures symptoms. The factor analysis model has three symptom components, three personality components and one component mixing both the personality and symptom variables. Table A.41 presents the ten component solution. With 16 variables and ten components the methods all have single indicator components. In all methods there is some degree of mixing of personality and symptoms in the components.

Post Treatment Results

The eigenvalues for the personality and symptoms covariance matrix is presented in Table A.42, for the post treatment depressed females. Like the results for baseline, seven components are needed to retain more than 90% of the variance.

The one component solutions are presented in Table A.43. Principal components and factor analysis lead to the same solution. The component measures self directedness versus harm avoidance and a weighted average of all the symptom variables. The independent component is a weighted average of the symptom variables only.

Table A.44 presents the four component solutions., Interestingly the factor analysis method has produced a first component that is an average of all bar one symptom variable. The other three components are mainly personality variables. The five component solution (see Table A.45) has similar components to the four component solution with some components representing a single variable. The factor solution has a redundant component. Moving to the six component solutions (Table A.46) the structure again is similar with some more splitting up of the variables.

The seven component solution is presented in Table A.47. Factor analysis has still managed to retain an initial factor that is an average of most of the symptom variables. The three methods present quite different solutions. Finally the eight component solutions are presented in Table A.48. For the principal components only one component (PC 5)

Variables	PC1	PC2	PC3	PC4	PC5	PC6	PC7
NS TCI	0.078	0.065	0.317	-0.088	0.034	0.126	-0.021
HA TCI	0.006	**0.486**	-0.020	0.027	0.145	-0.064	-0.133
RD TCI	0.064	0.080	0.007	0.086	-0.020	-0.032	-0.244
P TCI	0.120	**-0.445**	-0.133	0.067	0.002	-0.138	0.065
S TCI	-0.103	**-0.559**	-0.206	-0.129	0.035	-0.035	-0.130
C TCI	0.074	-0.118	-0.377	-0.067	-0.013	-0.033	-0.272
ST TCI	0.345	-0.019	0.372	0.109	-0.015	0.127	-0.040
S SCL	**0.944**	0.006	0.102	0.000	0.198	0.113	-0.054
OC SCL	0.321	0.102	0.202	0.226	0.085	**0.883**	0.067
IS SCL	0.272	**0.826**	0.085	0.250	0.247	0.048	0.294
D SCL	0.358	0.349	0.138	**0.761**	0.131	0.293	-0.059
A SCL	**0.717**	-0.140	0.041	0.561	0.183	0.147	-0.021
AH SCL	0.127	0.173	**0.949**	0.190	0.073	-0.065	0.080
PA SCL	0.256	0.254	0.109	0.117	**0.900**	0.086	0.146
PI SCL	0.326	0.303	0.219	0.314	0.142	-0.089	**0.788**
P SCL	**0.666**	0.358	0.157	0.294	0.038	0.159	0.112
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7
NS TCI	0.059	-0.116	0.045	0.072	-0.013	0.013	-0.100
HA TCI	0.005	-0.080	0.217	-0.208	0.094	0.119	-0.144
RD TCI	0.050	0.048	0.134	-0.110	0.040	0.107	-0.047
P TCI	-0.047	0.354	-0.204	0.158	-0.066	-0.172	0.191
S TCI	0.042	0.165	-0.092	0.130	0.050	-0.153	0.133
C TCI	-0.037	0.040	0.111	-0.038	0.044	0.039	0.006
ST TCI	0.055	-0.009	0.047	0.076	-0.064	0.046	-0.068
S SCL	-0.466	-0.097	**1.021**	0.734	-0.344	0.138	-0.732
OC SCL	0.351	**-1.250**	-0.153	0.647	-0.231	-0.269	0.696
IS SCL	-0.470	**-0.678**	0.479	-0.810	-0.058	0.287	-0.482
D SCL	0.516	0.683	0.066	**-1.029**	-0.004	0.351	0.743
A SCL	0.089	**1.049**	0.046	-0.015	-0.242	-0.091	0.539
AH SCL	**1.021**	0.117	-0.194	0.085	-0.279	0.190	-0.803
PA SCL	0.441	0.208	0.167	-0.069	0.773	**-1.315**	-0.049
PI SCL	-0.687	0.178	**-1.221**	0.393	-0.776	-0.642	0.292
P SCL	-0.296	-0.132	0.425	0.000	-0.362	0.354	-0.246
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7
NS TCI	0.131	-0.013	-0.245	-0.106	**0.764**	0.248	-0.035
HA TCI	0.019	0.136	**0.985**	0.058	-0.045	-0.079	0.012
RD TCI	0.029	0.018	0.018	**0.495**	0.027	0.091	0.034
P TCI	0.083	-0.136	-0.232	-0.010	**-0.625**	0.054	-0.074
S TCI	-0.066	-0.368	**-0.543**	0.274	-0.175	-0.173	-0.118
C TCI	0.029	-0.172	-0.085	**0.940**	-0.156	-0.221	-0.077
ST TCI	0.319	0.073	-0.179	0.238	0.016	**0.533**	0.050
S SCL	**0.847**	0.181	0.001	0.046	-0.063	0.118	-0.089
OC SCL	**0.508**	0.146	0.001	-0.029	0.141	0.129	0.361
IS SCL	0.197	**0.902**	0.275	0.048	0.157	0.061	0.204
D SCL	0.520	0.330	0.119	0.010	0.016	0.165	**0.761**
A SCL	**0.805**	0.108	-0.010	0.069	-0.070	0.110	0.232
AH SCL	0.139	0.231	0.120	-0.166	0.143	**0.717**	0.107
PA SCL	0.398	**0.477**	0.161	-0.013	0.085	0.053	0.035
PI SCL	0.249	**0.692**	0.014	-0.168	-0.034	0.246	0.069
P SCL	**0.615**	0.383	0.062	-0.015	0.141	0.194	0.199

Table A.40: Depressed females TCI and SCL at baseline, seven component solution.

Variables	PC1	PC2	PC3	PC4	PC5	PC6	PC7	PC8	PC9	PC10
NS TCI	-0.036	0.065	0.197	**-0.555**	0.069	-0.028	0.051	-0.010	0.100	-0.111
HA TCI	**0.503**	-0.008	-0.028	-0.071	-0.053	0.030	0.119	-0.071	-0.044	0.196
RD TCI	0.134	0.021	0.030	0.103	0.015	0.016	-0.033	-0.066	-0.017	**0.472**
P TCI	-0.295	0.110	0.063	**0.923**	-0.018	-0.047	0.014	-0.002	0.054	0.090
S TCI	**-0.546**	0.018	-0.205	0.108	-0.083	-0.062	0.021	-0.125	-0.182	0.007
C TCI	-0.077	0.089	-0.355	0.182	-0.046	-0.043	-0.031	-0.163	-0.013	0.321
ST TCI	-0.001	0.265	**0.404**	0.119	0.195	0.074	0.009	-0.017	0.224	0.071
S SCL	0.060	**0.937**	0.098	0.022	0.168	0.131	0.175	0.075	0.158	0.041
OC SCL	0.083	0.209	0.131	-0.119	**0.915**	0.226	0.103	0.050	0.125	0.015
IS SCL	**0.863**	0.132	0.097	-0.051	0.134	0.175	0.238	0.323	0.108	-0.014
D SCL	0.337	0.208	0.189	0.013	0.321	**0.803**	0.128	0.095	0.186	-0.011
A SCL	-0.097	**0.511**	0.052	-0.055	0.223	0.485	0.198	0.376	0.263	0.433
AH SCL	0.175	0.077	**0.946**	-0.180	0.037	0.089	0.087	0.129	0.029	0.035
PA SCL	0.277	0.204	0.095	-0.050	0.107	0.113	**0.906**	0.124	0.074	-0.062
PI SCL	0.357	0.155	0.265	0.047	0.049	0.130	0.172	**0.780**	0.145	-0.310
P SCL	0.289	0.360	0.165	-0.138	0.192	0.250	0.118	0.171	**0.770**	-0.064
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8	IC 9	IC 10
NS TCI	-0.053	0.229	0.120	0.129	-0.067	**-0.830**	0.158	0.161	0.527	0.501
HA TCI	0.123	-0.106	-0.291	-0.168	-0.148	0.326	0.139	0.080	0.141	0.103
RD TCI	0.257	-0.371	-0.297	-0.237	-0.248	**0.693**	0.364	-0.116	-0.176	-0.143
P TCI	0.081	-0.789	-0.173	-0.064	0.035	2.116	-0.669	-0.475	**-2.126**	-1.484
S TCI	0.009	0.053	0.135	0.127	0.319	-0.171	-0.002	0.017	0.063	-0.113
C TCI	0.058	-0.219	-0.179	-0.106	-0.084	0.340	0.126	-0.061	-0.102	-0.107
ST TCI	0.049	-0.164	-0.086	-0.035	-0.143	0.274	-0.141	-0.038	-0.465	-0.166
S SCL	-0.601	0.618	-0.918	-0.549	0.924	-0.778	-0.358	**1.221**	-0.264	-0.083
OC SCL	0.327	0.339	-0.088	0.139	-0.904	-0.266	0.226	0.045	0.058	**-1.322**
IS SCL	-0.108	0.218	-0.930	-0.660	-0.634	**1.266**	0.334	0.212	0.366	0.078
D SCL	0.393	0.537	0.262	-0.104	1.305	-0.343	**-1.724**	-0.608	0.471	0.275
A SCL	0.599	-0.847	-0.213	-0.345	0.112	0.334	**1.950**	-1.025	0.423	0.216
AH SCL	**1.012**	0.217	0.113	-0.129	-0.120	0.183	0.237	0.432	-0.646	0.423
PA SCL	0.197	-0.325	-0.134	**1.608**	0.258	0.073	-0.088	0.093	0.026	0.059
PI SCL	-0.830	0.986	**1.009**	0.233	0.509	-0.407	0.493	-0.395	-0.207	-0.396
P SCL	-0.663	-0.946	-0.088	0.376	**-1.541**	-0.473	-0.673	-0.859	-1.243	0.882
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8	FA 9	FA 10
NS TCI	0.121	0.025	**-0.444**	-0.387	0.022	-0.316	0.140	-0.094	-0.087	-0.064
HA TCI	0.028	0.182	-0.083	**0.938**	0.030	-0.012	-0.087	-0.263	-0.024	0.014
RD TCI	0.046	0.012	0.007	0.012	**0.992**	0.091	0.051	-0.007	-0.038	0.021
P TCI	0.047	-0.098	**0.979**	-0.111	0.022	0.049	0.077	0.054	-0.032	-0.064
S TCI	-0.096	-0.160	0.099	-0.304	0.008	0.281	0.011	**0.843**	-0.119	-0.095
C TCI	0.024	-0.062	0.055	0.077	0.368	**0.658**	0.152	0.292	-0.085	-0.049
ST TCI	0.272	0.028	0.045	-0.121	0.069	-0.083	**0.944**	0.019	0.029	0.072
S SCL	**0.839**	0.176	0.081	0.002	0.006	-0.022	0.139	0.003	0.017	-0.090
OC SCL	**0.520**	0.114	-0.122	-0.049	-0.008	-0.113	0.144	-0.089	-0.032	0.348
IS SCL	0.230	**0.788**	-0.160	0.165	0.054	-0.067	0.053	-0.300	0.300	0.237
D SCL	0.551	0.242	-0.023	0.078	0.036	-0.132	0.086	-0.117	0.120	**0.760**
A SCL	**0.833**	0.027	0.046	0.007	0.102	-0.020	0.057	0.028	0.139	0.209
AH SCL	0.173	0.149	-0.118	0.043	0.072	**-0.620**	0.302	-0.073	0.185	0.111
PA SCL	0.406	**0.520**	-0.039	0.131	-0.030	-0.161	0.009	0.029	0.119	0.037
PI SCL	0.271	0.403	0.016	-0.011	-0.086	-0.256	0.048	-0.146	**0.801**	0.074
P SCL	**0.638**	0.251	-0.130	-0.017	-0.040	-0.082	0.167	-0.242	0.211	0.193

Table A.41: Depressed females TCI and SCL at baseline, ten component solution.

	Eigenvalue	Proportion	Cumulative
1	1.361	0.638	0.638
2	0.180	0.084	0.722
3	0.121	0.057	0.778
4	0.108	0.051	0.829
5	0.081	0.038	0.867
6	0.053	0.025	0.891
7	0.051	0.024	0.915
8	0.037	0.017	0.933
9	0.036	0.017	0.949
10	0.026	0.012	0.962
11	0.024	0.011	0.973
12	0.017	0.008	0.981
13	0.016	0.008	0.989
14	0.013	0.006	0.995
15	0.006	0.003	0.998
16	0.005	0.003	1.000

Table A.42: Eigenvalues and variances for the symptoms and personality of the depressed females at six months.

Variables	PC 1	IC 1	FA 1
NS TCI	-0.128	0.015	-0.136
HA TCI	**0.405**	-0.056	**0.410**
RD TCI	-0.162	0.019	-0.163
P TCI	-0.067	0.013	-0.066
S TCI	**-0.486**	0.074	**-0.485**
C TCI	-0.075	0.006	-0.065
ST TCI	0.233	-0.030	0.237
S SCL	**0.673**	**-0.171**	**0.661**
OC SCL	**0.900**	**-0.384**	**0.888**
IS SCL	**0.823**	**-0.294**	**0.795**
D SCL	**0.958**	**-0.487**	**0.938**
A SCL	**0.903**	**-0.295**	**0.899**
AH SCL	**0.732**	**-0.253**	**0.683**
PA SCL	**0.588**	**-0.116**	**0.562**
PI SCL	**0.675**	**-0.218**	**0.640**
P SCL	**0.815**	**-0.152**	**0.815**

Table A.43: Depressed females TCI and SCL at six months, one component solution.

Variables	PC 1	PC 2	PC 3	PC 4	IC 1	IC 2	IC 3	IC 4
NS TCI	-0.016	-0.174	-0.024	-0.104	0.059	-0.096	0.201	-0.037
HA TCI	0.126	**0.683**	0.080	0.114	0.053	0.215	**-0.902**	0.368
RD TCI	-0.161	0.066	-0.270	0.067	-0.235	0.021	-0.284	-0.077
P TCI	-0.027	**-0.585**	0.269	0.111	-0.061	0.112	**1.411**	-0.522
S TCI	-0.188	**-0.541**	-0.323	-0.101	-0.238	-0.290	0.583	-0.391
C TCI	0.118	-0.088	-0.368	0.007	-0.139	-0.208	-0.008	-0.066
ST TCI	**0.366**	-0.170	0.089	-0.018	0.124	-0.274	0.441	0.000
S SCL	**0.738**	0.243	-0.023	0.131	0.086	**-0.838**	-0.013	0.359
OC SCL	**0.866**	0.345	0.106	0.243	0.155	**-1.191**	-0.091	**0.726**
IS SCL	0.309	**0.622**	0.545	0.401	0.172	1.283	**-1.251**	**0.723**
D SCL	**0.699**	0.240	0.425	0.451	-0.105	0.114	**0.732**	0.281
A SCL	**0.843**	0.239	0.116	0.352	-0.196	**-0.777**	0.264	0.243
AH SCL	0.401	0.075	-0.022	**0.896**	**-2.223**	0.530	0.040	**-1.118**
PA SCL	0.130	0.486	0.185	**0.555**	-0.555	0.676	**-0.832**	-0.004
PI SCL	0.333	-0.016	**0.838**	0.281	0.693	**0.931**	**1.464**	0.111
P SCL	0.555	0.287	**0.569**	0.226	0.380	0.154	0.207	0.333

Variables	FA 1	FA 2	FA 3	FA 4
NS TCI	-0.112	-0.164	**-0.567**	-0.024
HA TCI	0.310	-0.343	**0.551**	-0.382
RD TCI	-0.148	0.033	-0.022	**-0.342**
P TCI	-0.072	0.041	-0.043	**0.239**
S TCI	-0.347	**0.845**	-0.048	0.405
C TCI	0.066	**0.591**	0.060	-0.095
ST TCI	0.303	0.054	**-0.383**	0.110
S SCL	**0.714**	0.063	-0.053	-0.035
OC SCL	**0.918**	-0.073	-0.010	-0.114
IS SCL	**0.680**	-0.421	0.357	0.129
D SCL	**0.892**	-0.205	0.168	0.134
A SCL	**0.953**	-0.012	-0.015	-0.081
AH SCL	**0.682**	-0.029	0.076	0.048
PA SCL	**0.502**	-0.118	0.345	0.112
PI SCL	0.533	-0.505	-0.047	**0.624**
P SCL	**0.742**	-0.341	0.085	0.216

Table A.44: Depressed females TCI and SCL at six months, four component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
NS TCI	0.028	-0.363	-0.041	0.158	-0.182	0.409	0.037	-0.165	-0.318	**-0.577**
HA TCI	0.086	**0.735**	0.090	-0.067	-0.167	**-1.026**	-0.135	-0.123	0.565	0.264
RD TCI	-0.200	0.126	0.041	-0.356	0.054	**-0.451**	0.157	0.155	0.132	0.447
P TCI	-0.063	-0.101	-0.033	-0.005	**0.820**	0.819	-0.156	0.854	0.276	**1.929**
S TCI	-0.181	**-0.581**	-0.088	-0.237	0.129	**0.568**	0.255	0.314	-0.443	0.029
C TCI	0.087	-0.064	-0.009	**-0.420**	0.048	-0.113	0.099	0.294	0.003	0.161
ST TCI	0.393	-0.210	-0.002	0.151	0.002	**0.542**	-0.065	0.147	-0.199	-0.396
S SCL	**0.771**	0.033	0.193	0.096	-0.353	0.412	0.111	0.120	-0.425	**-1.768**
OC SCL	**0.860**	0.376	0.227	0.020	-0.082	-0.176	-0.172	**1.056**	0.461	-0.768
IS SCL	0.315	**0.709**	0.377	0.440	-0.069	-1.102	-0.167	**-1.343**	0.873	0.078
D SCL	**0.692**	0.472	0.380	0.238	0.236	0.295	-0.062	0.591	0.845	**1.201**
A SCL	**0.837**	0.320	0.322	0.012	0.028	0.114	0.156	**0.848**	0.189	-0.157
AH SCL	0.417	0.085	**0.892**	-0.024	0.058	0.131	**2.276**	-0.559	-1.097	0.401
PA SCL	0.149	0.403	**0.581**	0.213	-0.221	-0.610	0.621	**-0.930**	-0.107	-0.248
PI SCL	0.394	0.101	0.262	**0.842**	0.190	**1.986**	-0.451	-1.187	-0.249	-0.669
P SCL	**0.568**	0.436	0.184	0.456	0.104	0.237	-0.367	-0.117	**0.337**	-0.059

Variables	FA 1	FA 2	FA 3	FA 4	FA 5
NS TCI	-0.118	-0.156	0.069	**0.571**	0.039
HA TCI	0.292	-0.320	0.142	**-0.537**	-0.437
RD TCI	-0.109	0.138	**0.978**	0.114	-0.001
P TCI	-0.036	0.034	0.016	0.052	0.283
S TCI	-0.335	**0.811**	-0.212	0.005	0.431
C TCI	0.058	**0.620**	0.223	-0.047	-0.002
ST TCI	0.316	0.084	0.117	**0.408**	0.149
S SCL	**0.688**	0.088	-0.119	0.060	-0.168
OC SCL	**0.893**	-0.040	-0.032	0.039	-0.234
IS SCL	**0.723**	-0.402	0.066	-0.327	0.064
D SCL	**0.919**	-0.170	0.017	-0.135	0.033
A SCL	**0.934**	0.027	-0.059	0.047	-0.218
AH SCL	**0.693**	-0.001	0.046	-0.045	-0.006
PA SCL	**0.533**	-0.105	0.050	-0.323	0.073
PI SCL	**0.609**	-0.493	-0.198	0.046	0.475
P SCL	**0.772**	-0.335	-0.114	-0.061	0.104

Table A.45: Depressed females TCI and SCL at six months, five component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
NS TCI	-0.061	-0.328	0.210	-0.041	-0.247	0.060
HA TCI	0.230	**0.690**	-0.073	0.052	-0.189	-0.195
RD TCI	-0.086	0.078	-0.360	0.031	0.059	-0.201
P TCI	-0.037	-0.134	-0.072	-0.020	**0.939**	0.077
S TCI	-0.277	**-0.554**	-0.271	-0.035	0.203	0.155
C TCI	0.065	-0.083	**-0.478**	0.008	0.144	0.169
ST TCI	0.199	-0.148	0.068	0.045	0.134	**0.521**
S SCL	**0.597**	0.068	0.046	0.188	-0.273	**0.580**
OC SCL	**0.853**	0.315	0.046	0.126	-0.108	0.290
IS SCL	0.349	**0.735**	0.336	0.399	0.046	0.176
D SCL	**0.811**	0.373	0.306	0.241	0.148	0.016
A SCL	**0.863**	0.242	0.047	0.208	-0.004	0.244
AH SCL	0.606	-0.020	0.032	**0.780**	0.003	-0.077
PA SCL	0.161	0.451	0.067	**0.652**	-0.036	0.246
PI SCL	0.337	0.138	0.853	0.238	0.171	0.190
P SCL	**0.537**	0.439	0.424	0.152	0.143	0.267
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
NS TCI	0.036	0.495	-0.185	0.074	0.152	**-0.763**
HA TCI	0.217	-0.865	0.100	0.256	**-0.933**	-0.128
RD TCI	-0.172	-0.447	0.297	-0.081	-0.266	0.287
P TCI	-0.208	0.288	0.709	-0.788	0.794	**3.042**
S TCI	-0.410	0.312	0.024	-0.282	**1.018**	0.652
C TCI	-0.193	-0.259	0.112	-0.015	0.397	**0.576**
ST TCI	-0.099	0.255	-0.402	0.024	**1.136**	0.653
S SCL	-0.271	0.077	-1.012	0.635	**1.954**	-0.075
OC SCL	0.197	-0.240	0.377	**1.191**	0.594	0.069
IS SCL	-0.041	-1.322	**-1.643**	-0.141	-0.294	1.170
D SCL	0.288	0.637	1.281	0.284	**-1.634**	0.039
A SCL	-0.165	0.042	0.509	**0.571**	0.435	0.292
AH SCL	**-2.244**	0.280	0.169	-1.055	-0.242	-0.876
PA SCL	-0.862	-0.903	**-1.290**	-0.434	0.641	0.725
PI SCL	0.508	**2.046**	-1.163	-0.345	0.096	-0.637
P SCL	0.310	0.137	-0.354	0.145	0.142	**0.496**
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6
NS TCI	-0.090	-0.159	-0.129	0.076	**0.512**	0.067
HA TCI	0.287	-0.390	0.189	0.179	**-0.518**	-0.298
RD TCI	-0.112	0.055	-0.018	**0.846**	0.136	0.027
P TCI	-0.052	0.054	-0.017	0.014	0.058	**0.359**
S TCI	-0.298	**0.882**	-0.180	-0.179	0.008	0.262
C TCI	0.101	**0.599**	-0.053	0.329	-0.013	-0.052
ST TCI	0.281	0.128	0.101	0.100	**0.575**	0.019
S SCL	**0.694**	0.057	0.109	-0.120	0.077	-0.199
OC SCL	**0.888**	-0.095	0.189	-0.035	0.045	-0.190
IS SCL	0.526	-0.276	**0.763**	0.002	-0.142	-0.067
D SCL	**0.891**	-0.197	0.296	0.011	-0.163	0.229
A SCL	**0.927**	-0.048	0.152	-0.041	0.025	-0.132
AH SCL	**0.653**	-0.017	0.220	0.043	-0.027	0.044
PA SCL	0.396	-0.008	**0.521**	0.017	-0.127	-0.083
PI SCL	0.443	-0.345	**0.473**	-0.332	0.183	0.396
P SCL	**0.653**	-0.265	0.465	-0.180	0.075	0.066

Table A.46: Depressed females TCI and SCL at six months, six component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7
NS TCI	-0.104	-0.203	-0.217	-0.194	0.082	**0.417**	0.058
HA TCI	0.348	0.322	-0.140	-0.225	-0.151	**-0.563**	-0.093
RD TCI	-0.076	-0.009	0.048	0.054	**-0.427**	-0.096	0.029
P TCI	-0.037	-0.036	0.156	**0.940**	-0.038	0.094	0.000
S TCI	-0.416	-0.220	**0.577**	0.167	0.085	0.157	0.050
C TCI	0.053	0.010	**0.545**	0.091	-0.178	-0.039	-0.020
ST TCI	0.281	0.098	0.091	0.158	0.123	**0.538**	-0.055
S SCL	**0.630**	0.269	0.401	-0.322	0.405	0.117	0.074
OC SCL	**0.951**	0.181	-0.063	-0.107	0.028	0.113	0.108
IS SCL	0.545	**0.700**	-0.351	0.043	0.150	-0.183	0.054
D SCL	**0.811**	0.217	-0.135	0.126	0.299	-0.280	0.291
A SCL	**0.896**	0.179	0.067	-0.025	0.128	0.000	0.229
AH SCL	0.474	0.306	-0.020	-0.008	-0.017	0.058	**0.817**
PA SCL	0.261	**0.759**	0.037	-0.071	0.095	-0.098	0.269
PI SCL	0.355	0.260	-0.470	0.206	**0.693**	0.106	0.187
P SCL	**0.675**	0.358	-0.301	0.156	0.301	0.016	0.010
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7
NS TCI	-0.676	0.085	-0.280	0.159	-0.281	**-0.944**	-0.542
HA TCI	**1.150**	-0.313	0.742	-0.093	-0.276	0.310	-0.237
RD TCI	0.103	0.189	**0.529**	-0.314	0.011	-0.296	0.347
P TCI	-0.699	0.219	-0.239	-0.740	0.997	0.379	**3.008**
S TCI	-0.225	0.299	-0.614	0.037	0.715	**1.486**	0.344
C TCI	-0.031	0.138	0.123	-0.086	0.233	**0.776**	0.425
ST TCI	**-1.398**	0.206	-0.104	0.388	0.059	-0.135	0.768
S SCL	-0.662	0.113	-0.616	1.141	0.132	**2.783**	-0.590
OC SCL	**-1.647**	0.097	0.679	-0.425	-1.424	-1.144	0.468
IS SCL	0.160	-0.013	1.424	**1.607**	0.148	-0.171	1.227
D SCL	**2.271**	-0.452	-0.974	-1.240	-0.289	0.797	-0.234
A SCL	-0.380	0.212	-0.064	**-0.486**	-0.451	0.434	0.260
AH SCL	0.013	**2.264**	-0.084	-0.158	0.921	-1.069	-0.718
PA SCL	-0.008	0.710	0.707	**1.332**	0.818	1.111	0.493
PI SCL	-0.422	-0.447	**-1.928**	1.139	0.133	-0.899	-0.465
P SCL	-0.424	-0.249	-0.015	0.327	-0.210	-0.339	**0.608**
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7
NS TCI	-0.058	-0.073	-0.069	0.061	**0.678**	-0.033	0.035
HA TCI	0.252	-0.519	0.161	0.134	**-0.474**	-0.223	-0.156
RD TCI	-0.120	-0.056	-0.002	**0.789**	0.130	0.035	0.015
P TCI	-0.069	0.089	-0.047	0.022	0.001	**0.430**	0.031
S TCI	-0.260	**0.929**	-0.113	-0.045	-0.042	0.140	-0.048
C TCI	0.111	**0.512**	-0.069	0.462	-0.185	0.011	0.156
ST TCI	0.278	0.119	0.065	0.152	0.369	0.107	**0.472**
S SCL	**0.704**	0.019	0.115	-0.097	0.008	-0.202	0.117
OC SCL	**0.883**	-0.178	0.124	-0.045	-0.069	-0.093	0.182
IS SCL	**0.507**	-0.394	0.616	-0.066	-0.213	0.042	0.157
D SCL	**0.864**	-0.238	0.326	-0.028	-0.127	0.258	-0.091
A SCL	**0.920**	-0.114	0.131	-0.034	-0.066	-0.062	0.125
AH SCL	**0.651**	-0.026	0.354	0.032	0.066	-0.079	-0.119
PA SCL	0.370	-0.051	**0.669**	0.017	-0.119	-0.202	0.013
PI SCL	0.418	-0.271	**0.482**	-0.402	0.190	0.377	0.070
P SCL	**0.613**	-0.319	0.347	-0.235	-0.116	0.256	0.357

Table A.47: Depressed females TCI and SCL at six months, seven component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7	PC 8
NS TCI	-0.137	-0.272	-0.308	-0.154	0.042	0.090	0.379	0.024
HA TCI	0.341	0.295	-0.290	-0.134	-0.325	-0.099	**-0.494**	0.239
RD TCI	-0.081	-0.040	-0.017	0.090	**-0.488**	0.040	-0.075	-0.037
P TCI	-0.024	-0.051	0.118	**0.959**	-0.078	0.003	0.127	-0.088
S TCI	-0.405	-0.156	**0.743**	0.079	0.255	0.036	0.105	-0.095
C TCI	0.042	0.038	**0.604**	0.056	-0.130	-0.025	-0.016	0.067
ST TCI	0.228	0.043	0.027	0.166	0.096	-0.016	**0.569**	0.133
S SCL	0.516	0.133	0.062	-0.157	0.131	0.122	0.175	**0.786**
OC SCL	**0.931**	0.178	-0.050	-0.133	0.018	0.110	0.181	0.134
IS SCL	0.546	**0.702**	-0.380	0.050	0.088	0.055	-0.104	0.110
D SCL	**0.828**	0.233	-0.149	0.135	0.246	0.264	-0.233	0.165
A SCL	**0.878**	0.173	0.049	-0.028	0.090	0.224	0.058	0.216
AH SCL	0.469	0.269	-0.057	0.006	-0.045	**0.827**	0.062	0.094
PA SCL	0.261	**0.793**	0.112	-0.121	0.145	0.270	-0.054	0.080
PI SCL	0.368	0.258	-0.485	0.212	**0.673**	0.180	0.104	0.068
P SCL	**0.682**	0.377	-0.270	0.128	0.299	0.001	0.075	0.050
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8
NS TCI	0.345	-0.514	-0.006	-0.359	-0.724	0.754	-0.708	**-0.740**
HA TCI	-0.528	-0.451	0.406	-0.381	**1.321**	-0.354	-1.125	-0.579
RD TCI	-0.404	**-0.851**	-0.122	-0.395	0.224	0.034	-0.834	0.099
P TCI	0.357	-1.771	-0.159	-0.726	-0.527	-1.314	-1.158	**2.576**
S TCI	0.296	1.394	-0.491	0.826	-0.304	-1.106	**2.254**	0.937
C TCI	-0.213	0.329	-0.193	0.317	0.013	**-0.694**	0.563	0.538
ST TCI	0.054	-0.174	-0.161	-0.130	**-1.471**	-0.183	-0.494	0.577
S SCL	0.755	0.102	0.104	-0.803	-0.448	**-3.166**	-1.760	-1.499
OC SCL	-0.848	-0.320	-0.132	1.529	**-1.617**	1.317	-0.135	0.401
IS SCL	-1.315	0.953	0.117	-0.922	-0.148	-0.051	**-1.345**	1.042
D SCL	1.049	-0.224	0.348	0.808	**2.491**	-0.380	0.647	-0.090
A SCL	-0.044	-0.064	-0.267	**0.933**	-0.259	-0.272	0.358	0.255
AH SCL	0.174	-0.758	**-2.214**	-1.362	-0.050	0.717	-0.052	-0.645
PA SCL	-0.925	**2.153**	-0.806	-0.094	-0.320	-0.833	1.279	0.976
PI SCL	**1.912**	0.843	0.462	-0.544	-0.867	0.849	0.027	-0.322
P SCL	-0.040	0.337	0.233	0.250	-0.563	0.346	-0.054	**0.653**
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8
NS TCI	-0.068	-0.291	-0.102	**-0.407**	0.339	0.153	-0.092	-0.207
HA TCI	0.217	-0.215	0.142	**0.906**	-0.069	0.066	-0.237	-0.034
RD TCI	-0.110	0.100	-0.014	0.007	0.087	**0.810**	0.020	-0.021
P TCI	-0.067	0.073	-0.045	-0.075	0.053	0.016	**0.452**	0.012
S TCI	-0.318	**0.753**	-0.099	-0.317	-0.024	-0.213	0.169	-0.166
C TCI	0.073	**0.690**	-0.037	0.020	0.092	0.270	0.050	0.058
ST TCI	0.222	0.096	0.052	-0.093	**0.950**	0.086	0.139	0.034
S SCL	**0.683**	0.054	0.106	0.071	0.167	-0.156	-0.213	-0.013
OC SCL	**0.897**	-0.081	0.109	0.116	0.104	-0.040	-0.109	0.149
IS SCL	0.547	-0.279	**0.614**	0.274	-0.015	-0.011	0.046	0.224
D SCL	**0.878**	-0.158	0.291	0.217	-0.041	-0.013	0.252	-0.080
A SCL	**0.924**	-0.014	0.121	0.104	0.090	-0.049	-0.067	0.073
AH SCL	**0.660**	-0.014	0.342	-0.041	-0.024	0.056	-0.082	-0.158
PA SCL	0.379	0.016	**0.664**	0.132	0.026	-0.003	-0.166	-0.018
PI SCL	**0.444**	-0.421	0.444	-0.068	0.125	-0.301	0.352	-0.024
P SCL	**0.653**	-0.271	0.336	0.121	0.131	-0.186	0.251	0.341

Table A.48: Depressed females TCI and SCL at six months, eight component solution.

	Eigenvalue	Proportion	Cumulative
1	2.766	0.516	0.516
2	0.630	0.118	0.633
3	0.497	0.093	0.726
4	0.349	0.065	0.791
5	0.300	0.056	0.847
6	0.236	0.044	0.891
7	0.154	0.029	0.919
8	0.142	0.027	0.946
9	0.102	0.019	0.965
10	0.071	0.013	0.978
11	0.044	0.008	0.986
12	0.023	0.004	0.990
13	0.018	0.003	0.994
14	0.016	0.003	0.997
15	0.011	0.002	0.999
16	0.007	0.001	1.000

Table A.49: Eigenvalues and variances for the symptoms and personality of the depressed males at baseline.

has a mixture of personality and symptom variables. Factor analysis has one redundant component and no mixing of personality and symptoms. In comparison ICA has five of the eight components with both personality and symptom variables.

Comparison Across Time

Two, three, four, seven and ten components were retained for the personality and symptom components of the baseline depressed females. In comparison, after treatment one, four, five, six, seven and eight components were retained. The four component solution at baseline is presented in Table A.39 and the after treatment four component solution is presented in Table A.44. The solutions are different after treatment compared to the baseline solutions. Likewise the seven component solutions are different across time.

A.9 Depressed Males Personality and Symptoms

Baseline Results

As with the depressed females, seven components are needed to retain approximately 90% of the variance (Table A.49). The one component solution is presented in Table A.50. The principal components and factors both have personality and symptoms with high loadings. The independent component only has high loadings from the symptom variables.

Table A.51 presents the three component solution. The independent component solution has one redundant component and no contributions from personality in the other

Variables	PC 1	IC 1	FA 1
NS TCI	-0.018	-0.001	-0.067
HA TCI	0.384	0.032	**0.450**
RD TCI	0.047	0.002	0.004
P TCI	0.148	0.014	0.138
S TCI	**-0.507**	-0.034	**-0.571**
C TCI	**-0.419**	-0.021	**-0.468**
ST TCI	0.214	0.014	0.205
S SCL	**0.606**	**0.138**	**0.581**
OC SCL	**0.699**	**0.193**	**0.663**
IS SCL	**0.849**	**0.261**	**0.795**
D SCL	**0.707**	**0.179**	**0.677**
A SCL	**0.744**	**0.196**	**0.722**
AH SCL	**0.487**	**0.141**	**0.419**
PA SCL	**0.760**	**0.212**	**0.716**
PI SCL	**0.800**	**0.266**	**0.708**
P SCL	**0.816**	**0.181**	**0.838**

Table A.50: Depressed males TCI and SCL at baseline, one component solution.

Variables	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
NS TCI	-0.089	0.056	0.150	0.007	0.045	0.036	0.049	0.065	**0.394**
HA TCI	0.372	0.082	0.077	-0.035	-0.020	-0.011	0.186	0.106	**-0.709**
RD TCI	-0.017	0.188	0.022	0.012	0.001	0.047	0.173	-0.039	0.170
P TCI	0.078	0.081	0.172	-0.008	0.058	0.045	0.083	0.089	-0.105
S TCI	**-0.484**	-0.106	-0.123	0.037	0.014	0.010	-0.251	-0.194	**0.769**
C TCI	-0.306	-0.022	**-0.443**	0.028	-0.079	-0.011	-0.122	-0.288	**0.526**
ST TCI	0.186	0.085	0.066	-0.011	-0.002	0.011	0.172	0.252	0.066
S SCL	0.375	**0.558**	0.331	0.002	0.139	**0.540**	**0.614**	0.301	0.029
OC SCL	**0.599**	0.426	0.100	-0.091	-0.187	**0.299**	**0.542**	0.272	-0.298
IS SCL	**0.923**	-0.068	0.113	-0.402	-0.241	**-0.513**	0.395	**0.626**	-0.387
D SCL	**0.737**	0.281	-0.141	-0.145	**-0.474**	-0.035	**0.558**	0.325	-0.220
A SCL	0.540	**0.724**	0.134	0.017	-0.162	**0.678**	**0.882**	0.167	-0.100
AH SCL	0.210	0.033	**0.938**	-0.178	**1.137**	0.326	0.179	**0.380**	-0.193
PA SCL	**0.640**	0.473	0.132	-0.096	-0.173	**0.357**	**0.666**	0.299	-0.120
PI SCL	**0.840**	-0.230	0.355	-0.492	0.177	**-0.640**	0.220	**0.968**	-0.117
P SCL	**0.719**	0.359	0.196	-0.133	-0.083	**0.159**	**0.615**	0.431	-0.301

Table A.51: Depressed males TCI and SCL at baseline, three component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
NS TCI	-0.193	0.167	0.073	0.009	-0.047	0.009	-0.009	0.011	0.021	-0.107
HA TCI	0.333	0.056	0.154	0.185	0.051	-0.063	0.034	0.020	-0.025	0.091
RD TCI	0.024	-0.004	0.028	0.017	0.247	0.009	0.055	-0.009	0.043	0.040
P TCI	0.086	-0.022	0.208	0.033	0.148	-0.009	0.135	0.025	0.008	0.072
S TCI	**-0.505**	-0.072	-0.234	-0.075	-0.179	-0.033	-0.066	-0.031	-0.007	-0.145
C TCI	-0.170	-0.196	**-0.465**	-0.084	0.024	-0.004	-0.057	-0.052	0.020	0.030
ST TCI	0.078	0.283	0.022	0.035	-0.052	0.046	-0.064	0.016	0.036	-0.080
S SCL	0.039	**0.909**	0.143	0.148	0.160	0.471	-0.303	0.118	0.681	**-0.912**
OC SCL	**0.686**	0.226	0.208	0.005	0.578	0.621	0.500	0.084	0.566	**0.934**
IS SCL	**0.790**	0.179	0.280	0.354	-0.215	-0.420	-0.268	0.247	**-0.502**	0.473
D SCL	**0.718**	0.259	-0.033	0.237	0.200	0.063	-0.181	-0.008	0.206	**0.567**
A SCL	0.313	**0.593**	0.086	0.466	0.409	-0.192	0.040	-0.049	**0.658**	-0.221
AH SCL	0.034	0.121	**0.973**	0.083	0.094	-0.096	**1.168**	0.519	-0.239	-0.217
PA SCL	0.346	0.297	0.181	**0.818**	0.065	**-1.336**	0.031	-0.060	-0.067	-0.160
PI SCL	**0.626**	0.486	0.411	0.001	-0.403	0.620	**-0.632**	0.557	-0.363	-0.314
P SCL	0.480	**0.562**	0.198	0.343	0.066	-0.020	-0.193	0.111	0.214	-0.190

Variables	FA 1	FA 2	FA 3	FA 4	FA 5
NS TCI	0.098	-0.048	**0.595**	0.031	-0.242
HA TCI	0.238	-0.232	**-0.645**	0.091	0.055
RD TCI	0.121	**0.370**	0.044	-0.027	0.218
P TCI	0.034	0.015	-0.209	0.000	**0.442**
S TCI	-0.334	0.302	**0.596**	-0.160	-0.028
C TCI	-0.207	**0.932**	0.252	-0.063	0.020
ST TCI	0.127	0.044	-0.033	0.101	**0.600**
S SCL	**0.657**	-0.187	0.202	0.078	0.233
OC SCL	**0.587**	-0.062	-0.250	0.191	0.105
IS SCL	0.539	-0.118	-0.346	**0.635**	-0.100
D SCL	**0.626**	0.112	-0.234	0.318	-0.013
A SCL	**0.880**	-0.006	-0.072	-0.009	0.193
AH SCL	0.248	**-0.433**	-0.009	0.221	0.138
PA SCL	**0.714**	-0.066	-0.089	0.195	0.016
PI SCL	0.370	-0.266	0.024	**0.852**	0.255
P SCL	**0.719**	-0.304	-0.114	0.280	0.021

Table A.52: Depressed males TCI and SCL at baseline, five component solution.

two components. The PC model has two components mixing personality and symptoms, and one symptom component. The factor solution has two symptom factors and one personality factor. This personality factor is the same as the baseline personality factor found when looking at personality alone.

Table A.52 presents the five component solutions. The PC's and IC's have one redundant factor each. The IC solution has no contributions from personality. The factor analysis solution has two personality factors, two symptom factors and one mixed factor.

The six component solutions are presented in Table A.53. The PC and IC solutions are dominated by the symptoms, however factor analysis has contributions from all the variables. The seven component solutions from PCA and FA both have one redundant component. In fact the FA solution is the same as the FA six component solution. The independent component solution has two redundant components.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6
NS TCI	-0.127	0.189	0.084	-0.013	-0.113	-0.036
HA TCI	0.292	0.017	0.140	0.192	0.145	0.119
RD TCI	0.054	0.017	0.034	0.007	0.177	-0.165
P TCI	0.005	-0.010	0.189	0.067	0.204	-0.009
S TCI	-0.389	-0.016	-0.200	-0.108	-0.352	-0.162
C TCI	-0.080	-0.174	**-0.437**	-0.115	-0.121	-0.200
ST TCI	0.027	0.271	0.001	0.057	0.032	0.143
S SCL	0.151	**0.912**	0.137	0.108	0.110	0.031
OC SCL	0.337	0.208	0.116	0.142	**0.888**	0.130
IS SCL	0.547	0.061	0.214	0.430	0.191	**0.581**
D SCL	**0.946**	0.133	0.001	0.115	0.148	0.046
A SCL	0.511	**0.585**	0.108	0.384	0.256	-0.174
AH SCL	0.108	0.112	**0.982**	0.043	0.089	0.024
PA SCL	0.277	0.303	0.156	**0.851**	0.152	0.105
PI SCL	0.365	0.354	0.331	0.084	0.075	**0.763**
P SCL	0.486	**0.500**	0.176	0.330	0.181	0.226
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6
NS TCI	0.013	0.010	-0.056	-0.059	0.030	0.067
HA TCI	-0.026	-0.067	0.052	0.080	0.009	-0.046
RD TCI	-0.052	0.011	0.035	0.034	-0.031	0.023
P TCI	-0.166	0.007	0.068	-0.018	-0.006	-0.035
S TCI	0.061	-0.033	-0.109	-0.096	0.010	0.047
C TCI	0.063	-0.009	0.002	0.029	-0.061	-0.005
ST TCI	0.044	0.051	-0.013	-0.111	0.006	0.041
S SCL	0.324	0.482	-0.213	-0.635	0.044	**0.927**
OC SCL	-0.850	0.751	**1.042**	-0.336	-0.543	-0.229
IS SCL	0.150	-0.425	0.289	0.004	0.213	**-0.682**
D SCL	0.687	-0.109	0.251	**1.458**	-0.042	0.300
A SCL	0.106	-0.224	0.046	0.184	-0.191	**0.741**
AH SCL	**-0.881**	-0.118	-0.188	0.645	0.803	0.321
PA SCL	-0.343	**-1.257**	-0.057	-0.681	-0.109	-0.184
PI SCL	0.576	0.608	0.020	-0.504	**0.621**	-0.210
P SCL	0.234	-0.038	0.026	-0.075	0.055	0.282
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6
NS TCI	0.096	**0.612**	0.021	-0.182	-0.346	0.102
HA TCI	0.208	**-0.667**	0.090	-0.197	0.064	-0.075
RD TCI	0.073	0.071	-0.027	0.059	0.129	**0.646**
P TCI	0.016	-0.194	-0.008	-0.088	**0.405**	0.187
S TCI	-0.282	**0.632**	-0.158	0.356	0.029	-0.042
C TCI	-0.178	0.324	-0.048	**0.753**	0.033	0.426
ST TCI	0.148	-0.013	0.089	0.021	**0.631**	0.028
S SCL	**0.665**	0.170	0.060	-0.242	0.216	-0.070
OC SCL	**0.545**	-0.274	0.196	-0.211	0.035	0.246
IS SCL	0.513	-0.360	**0.644**	-0.119	-0.076	-0.046
D SCL	**0.610**	-0.244	0.340	0.050	-0.029	0.144
A SCL	**0.863**	-0.101	0.003	-0.093	0.162	0.122
AH SCL	0.192	-0.039	0.190	**-0.582**	0.056	0.075
PA SCL	**0.707**	-0.108	0.201	-0.095	0.018	0.015
PI SCL	0.349	0.011	**0.837**	-0.335	0.248	-0.056
P SCL	**0.719**	-0.158	0.274	-0.281	0.022	-0.172

Table A.53: Depressed males TCI and SCL at baseline, six component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7
NS TCI	0.102	-0.283	0.065	0.062	0.015	0.058	-0.041
HA TCI	0.145	**0.461**	0.194	0.013	0.145	-0.163	0.090
RD TCI	0.032	-0.071	-0.032	-0.042	0.012	0.364	0.081
P TCI	-0.031	-0.034	0.116	0.091	0.075	0.379	-0.004
S TCI	-0.166	**-0.584**	-0.225	-0.097	-0.049	-0.029	-0.126
C TCI	-0.201	-0.257	**-0.474**	-0.151	-0.081	0.089	0.084
ST TCI	0.180	-0.153	-0.042	0.287	0.082	0.166	0.022
S SCL	**0.903**	-0.045	0.171	0.240	0.074	-0.018	0.010
OC SCL	0.384	**0.654**	0.044	0.217	0.039	0.609	-0.032
IS SCL	0.126	**0.648**	0.245	0.500	0.410	-0.138	0.211
D SCL	0.249	0.373	-0.031	0.241	0.150	0.226	**0.803**
A SCL	**0.730**	0.241	0.110	0.015	0.345	0.187	0.323
AH SCL	0.092	0.041	**0.961**	0.100	0.054	0.223	0.057
PA SCL	0.338	0.138	0.107	0.221	**0.850**	0.248	0.114
PI SCL	0.198	0.182	0.307	**0.884**	0.128	0.034	0.160
P SCL	**0.571**	0.358	0.205	0.316	0.302	-0.031	0.233
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7
NS TCI	-0.059	-0.024	0.009	0.119	-0.087	0.002	-0.144
HA TCI	0.321	0.065	-0.027	-0.363	0.167	-0.095	0.283
RD TCI	-0.165	-0.085	-0.016	0.137	-0.014	0.110	-0.114
P TCI	-0.365	-0.228	-0.049	0.249	-0.050	0.103	-0.189
S TCI	-0.149	-0.010	-0.044	0.261	-0.180	0.023	-0.200
C TCI	-0.150	0.026	-0.035	0.172	-0.048	0.112	-0.082
ST TCI	-0.175	-0.010	0.031	0.229	-0.078	-0.006	-0.184
S SCL	**0.856**	0.295	0.682	0.169	0.113	-0.454	-0.562
OC SCL	-0.652	-0.772	0.623	0.353	**1.124**	-0.138	0.263
IS SCL	0.430	0.401	-0.409	**-0.979**	0.363	-0.614	0.978
D SCL	-0.509	0.654	-0.279	-0.165	-0.019	**1.522**	-0.492
A SCL	**0.668**	0.107	-0.112	-0.025	0.290	0.308	-0.335
AH SCL	0.032	-0.843	-0.107	**-0.924**	-0.300	0.323	-0.468
PA SCL	-0.356	-0.553	**-1.282**	0.906	-0.278	-0.374	-0.282
PI SCL	**-0.997**	0.447	0.458	0.384	-0.458	-0.437	-0.666
P SCL	**0.607**	0.314	0.060	-0.330	0.215	-0.208	0.049
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7
NS TCI	0.117	**0.407**	0.073	0.209	-0.524	0.149	-0.164
HA TCI	0.172	**-0.658**	0.038	0.082	0.182	-0.157	0.110
RD TCI	0.075	0.056	-0.025	0.000	0.118	**0.609**	-0.025
P TCI	0.016	-0.115	-0.021	0.175	**0.481**	0.193	0.040
S TCI	-0.228	**0.819**	-0.131	-0.194	-0.013	-0.005	0.024
C TCI	-0.192	0.420	-0.060	**-0.601**	0.012	0.531	0.091
ST TCI	0.186	0.045	0.149	-0.013	**0.554**	0.056	-0.091
S SCL	**0.699**	0.045	0.125	0.169	0.076	-0.059	-0.178
OC SCL	**0.524**	-0.370	0.169	0.157	0.051	0.212	0.062
IS SCL	0.470	-0.382	**0.555**	0.120	-0.031	-0.083	0.550
D SCL	**0.582**	-0.310	0.299	-0.108	-0.006	0.154	0.098
A SCL	**0.851**	-0.153	-0.003	0.079	0.137	0.129	0.054
AH SCL	0.193	-0.103	0.170	**0.667**	0.062	0.033	0.052
PA SCL	**0.699**	-0.077	0.134	0.166	0.075	0.011	0.279
PI SCL	0.372	-0.096	**0.858**	0.289	0.164	-0.064	0.027
P SCL	**0.728**	-0.280	0.273	0.167	-0.023	-0.188	-0.019

Table A.54: Depressed males TCI and SCL at baseline, seven component solution.

	Eigenvalue	Proportion	Cumulative
1	2.800	0.692	0.692
2	0.319	0.079	0.771
3	0.218	0.054	0.825
4	0.197	0.049	0.874
5	0.118	0.029	0.903
6	0.097	0.024	0.927
7	0.066	0.016	0.944
8	0.054	0.013	0.957
9	0.045	0.011	0.968
10	0.035	0.009	0.977
11	0.032	0.008	0.984
12	0.021	0.005	0.989
13	0.015	0.004	0.993
14	0.014	0.003	0.997
15	0.009	0.002	0.999
16	0.005	0.001	1.000

Table A.55: Eigenvalues and variances for the symptoms and personality of the depressed males at six months.

Post Treatment Results

For the post treatment depressed males, only five components need to be retained to keep 90% of the variance (Table A.55). The two component solutions are presented in Table A.56. All three methods have a symptom component and a mixed personality and symptom component.

Table A.57 presents the three component solutions. PCA and ICA have two symptom components and one mixed component. Factor analysis has two mixed components and a symptom component. The five component solutions are presented in Table A.58. The ICA solution has one redundant component and the factor analysis solution has two. The three components from factor analysis are different from the three component solution.

Table A.59 presents the eight component solution. The PC and IC solutions have two redundant components but factor analysis retains all eight components. Five of these components have single variable indicators. The first component is an average of seven of the symptom variables.

The nine component solution (Table A.60) has three redundant components for the IC and PC solutions and the factor analysis solution has one redundant component. Table A.61 presents the twelve component solution. For factor analysis SAS (SAS(R) Proprietary Software Release (8.1)) would only rotate 10 components and the solution is the same as the nine component solution. The PC solution has eight components with high loadings and the IC solution retains nine components.

Variables	PC 1	PC 2	IC 1	IC 2	FA 1	FA 2
NS TCI	-0.171	-0.030	-0.046	-0.012	-0.176	0.003
HA TCI	0.193	**0.592**	**-0.271**	0.033	0.165	**0.664**
RD TCI	-0.080	-0.312	0.075	-0.007	-0.092	**-0.336**
P TCI	0.144	-0.289	**0.240**	0.010	0.280	**-0.489**
S TCI	-0.248	**-0.594**	0.176	-0.028	-0.227	**-0.709**
C TCI	-0.076	**-0.573**	0.186	-0.011	-0.092	**-0.663**
ST TCI	0.380	-0.129	**0.227**	0.031	**0.345**	-0.139
S SCL	**0.768**	0.286	**0.474**	0.177	**0.836**	0.169
OC SCL	**0.751**	0.472	**0.311**	0.247	**0.661**	0.492
IS SCL	0.537	**0.797**	**-0.642**	0.232	0.606	**0.712**
D SCL	**0.706**	0.597	0.039	**0.264**	**0.743**	0.506
A SCL	0.601	**0.681**	**-0.251**	0.203	**0.714**	0.552
AH SCL	**0.848**	0.062	**1.027**	0.229	**0.591**	0.241
PA SCL	0.177	**0.887**	**-0.984**	0.076	0.342	**0.728**
PI SCL	**0.744**	0.418	**0.317**	0.195	**0.799**	0.318
P SCL	**0.725**	0.522	0.119	0.151	**0.851**	0.384

Table A.56: Depressed males TCI and SCL at six months, two component solution.

Variables	PC 1	PC 2	PC 3	IC 1	IC 2	IC 3	FA 1	FA 2	FA 3
NS TCI	-0.014	-0.187	-0.037	0.062	-0.040	0.019	-0.071	-0.015	**-0.329**
HA TCI	**0.594**	0.185	0.068	**0.284**	0.023	0.012	0.204	**0.684**	0.251
RD TCI	-0.296	-0.131	0.044	-0.059	-0.056	-0.005	-0.152	**-0.322**	-0.036
P TCI	-0.324	0.201	-0.023	**-0.301**	0.130	-0.048	0.191	**-0.547**	0.022
S TCI	**-0.582**	-0.270	-0.046	-0.165	-0.068	-0.001	-0.389	**-0.657**	0.064
C TCI	**-0.607**	0.004	-0.123	**-0.230**	0.082	-0.019	-0.344	**-0.596**	0.353
ST TCI	-0.146	0.354	0.165	**-0.243**	0.022	-0.068	0.214	-0.148	**0.301**
S SCL	0.192	**0.917**	0.062	-0.801	**0.861**	-0.248	**0.795**	0.030	0.284
OC SCL	0.474	**0.638**	0.395	-0.220	-0.170	**-0.295**	0.583	0.442	**0.614**
IS SCL	**0.805**	0.461	0.271	**0.766**	-0.121	-0.125	**0.706**	0.576	0.183
D SCL	0.548	**0.754**	0.169	-0.261	**0.693**	-0.265	**0.750**	0.376	0.353
A SCL	**0.651**	0.617	0.173	0.151	**0.391**	-0.159	**0.812**	0.394	0.081
AH SCL	0.167	0.394	**0.880**	-0.400	**-1.680**	-0.398	**0.548**	0.158	0.326
PA SCL	**0.920**	0.098	0.150	**1.150**	-0.251	0.083	0.520	**0.616**	-0.078
PI SCL	0.424	**0.616**	0.414	-0.222	-0.207	**-0.245**	**0.825**	0.153	0.209
P SCL	0.480	**0.743**	0.214	-0.218	**0.308**	-0.168	**0.941**	0.180	0.107

Table A.57: Depressed males TCI and SCL at six months, three component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	IC 1	IC 2	IC 3	IC 4	IC 5
NS TCI	0.026	-0.037	-0.017	-0.356	-0.056	0.095	0.144	0.288	-0.035	0.075
HA TCI	**0.487**	0.192	0.064	0.406	-0.236	0.146	-0.197	-0.227	0.389	0.150
RD TCI	-0.240	-0.123	0.048	-0.228	0.082	-0.026	0.100	0.075	-0.059	-0.026
P TCI	-0.199	0.027	-0.039	-0.151	**0.493**	-0.144	-0.027	-0.050	**-0.575**	-0.302
S TCI	**-0.525**	-0.322	-0.075	-0.182	0.109	-0.118	0.071	-0.124	-0.144	-0.112
C TCI	**-0.617**	-0.090	-0.108	0.077	0.109	-0.244	-0.110	-0.128	-0.023	-0.082
ST TCI	-0.090	0.125	0.160	0.164	0.366	-0.182	-0.039	-0.197	-0.212	-0.226
S SCL	0.217	**0.733**	0.156	0.237	0.491	**-0.695**	-0.577	0.569	-0.582	-0.378
OC SCL	0.402	0.377	0.402	**0.691**	0.101	-0.452	-0.445	**-1.353**	0.777	-0.379
IS SCL	**0.775**	0.359	0.261	0.375	0.044	**0.700**	-0.163	-0.456	0.304	-0.021
D SCL	0.466	**0.704**	0.269	0.405	0.052	-0.537	-0.613	**0.871**	0.851	0.253
A SCL	**0.672**	0.620	0.246	0.032	0.197	0.281	0.095	**1.466**	-0.237	0.129
AH SCL	0.234	0.218	**0.926**	0.081	0.174	-0.194	**1.823**	0.550	0.688	-0.306
PA SCL	**0.941**	0.122	0.093	0.053	-0.033	**1.252**	0.209	-0.042	-0.217	0.146
PI SCL	0.557	0.212	0.337	0.299	**0.641**	0.208	0.050	-1.217	**-1.362**	-1.077
P SCL	0.540	**0.546**	0.242	0.204	0.451	-0.045	-0.162	0.161	**-0.599**	-0.360

Variables	FA 1	FA 2	FA 3	FA 4	FA 5
NS TCI	-0.087	-0.040	**-0.495**	-0.037	-0.051
HA TCI	0.186	-0.709	**0.676**	-0.007	-0.078
RD TCI	-0.192	0.288	0.041	-0.037	-0.070
P TCI	0.126	**0.526**	0.036	-0.084	-0.151
S TCI	-0.356	**0.760**	-0.085	0.221	0.082
C TCI	-0.306	**0.673**	0.291	-0.035	0.230
ST TCI	0.223	0.195	**0.390**	-0.180	-0.113
S SCL	**0.796**	-0.027	0.171	-0.349	0.039
OC SCL	**0.726**	-0.258	0.403	0.077	0.147
IS SCL	**0.795**	-0.460	0.125	0.245	0.055
D SCL	**0.847**	-0.292	0.181	-0.053	0.401
A SCL	**0.808**	-0.373	0.127	-0.069	-0.042
AH SCL	**0.641**	-0.024	0.114	0.075	-0.029
PA SCL	**0.563**	-0.528	0.036	0.332	-0.099
PI SCL	**0.880**	-0.040	0.120	0.045	-0.248
P SCL	**0.917**	-0.180	0.062	-0.137	-0.084

Table A.58: Depressed males TCI and SCL at six months, five component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7	PC 8
NS TCI	0.012	-0.023	0.016	-0.008	**-0.645**	-0.213	0.024	-0.056
HA TCI	0.112	**0.803**	0.212	0.063	0.242	0.198	-0.022	-0.096
RD TCI	-0.120	-0.221	-0.114	0.045	0.009	-0.239	-0.024	-0.019
P TCI	0.134	-0.589	0.096	-0.060	**0.662**	-0.209	-0.040	0.037
S TCI	-0.346	**-0.696**	-0.167	-0.072	0.155	-0.081	-0.029	-0.086
C TCI	-0.152	**-0.469**	-0.382	-0.116	0.187	0.045	-0.050	-0.270
ST TCI	0.240	0.064	-0.106	0.135	**0.513**	-0.152	0.103	-0.149
S SCL	**0.934**	0.147	-0.010	0.119	0.080	0.090	0.041	-0.102
OC SCL	0.484	0.394	0.152	0.390	0.264	**0.545**	0.144	-0.189
IS SCL	**0.498**	0.421	0.440	0.261	0.072	0.416	0.123	0.334
D SCL	**0.716**	0.210	0.235	0.254	0.062	0.524	-0.153	0.090
A SCL	**0.717**	0.387	0.483	0.226	0.127	0.016	-0.130	0.040
AH SCL	0.357	0.072	0.126	**0.916**	0.060	0.045	0.051	0.030
PA SCL	0.261	0.308	**0.868**	0.107	-0.087	0.221	0.067	-0.007
PI SCL	**0.683**	0.080	0.333	0.321	0.130	0.118	0.508	0.049
P SCL	**0.811**	0.153	0.298	0.219	0.142	0.141	0.141	0.170
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8
NS TCI	0.044	-0.044	-0.071	-0.445	0.010	-0.744	-0.572	**-1.369**
HA TCI	-0.109	0.284	-0.412	1.108	0.172	-0.606	**1.427**	0.253
RD TCI	0.034	0.016	0.068	-0.028	0.085	0.136	0.029	0.026
P TCI	0.062	-0.158	0.241	-0.325	0.305	1.959	0.567	**2.313**
S TCI	0.379	-0.176	0.109	-0.701	-0.174	**0.827**	-0.339	0.795
C TCI	0.234	0.109	-0.076	**-0.505**	-0.156	0.241	-0.148	0.443
ST TCI	-0.181	0.296	0.097	0.309	0.184	0.341	**1.021**	0.875
S SCL	-0.733	0.862	-0.204	-0.704	0.520	**-0.939**	-0.147	-0.834
OC SCL	0.857	0.498	-0.393	-0.619	-1.066	**-1.068**	0.803	0.864
IS SCL	-1.113	-0.757	0.852	**2.012**	-0.315	0.236	-0.348	0.543
D SCL	0.658	0.165	-0.551	-0.484	0.271	0.285	**-2.068**	0.503
A SCL	0.088	0.147	-0.645	0.409	**1.762**	0.313	1.217	0.087
AH SCL	**1.386**	0.214	1.115	0.404	0.526	0.347	-0.009	-0.688
PA SCL	1.162	-1.397	-0.760	**-1.886**	-0.154	0.033	0.049	-0.375
PI SCL	-1.068	-0.380	1.145	**-1.221**	-0.900	-0.442	0.330	-0.389
P SCL	**-0.783**	-0.003	0.431	0.026	0.276	0.253	-0.057	0.175
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8
NS TCI	-0.066	-0.039	-0.181	0.014	**0.940**	-0.064	0.065	-0.044
HA TCI	0.300	-0.351	**-0.535**	0.312	-0.498	0.204	0.025	-0.097
RD TCI	-0.139	0.160	0.081	-0.068	0.063	-0.032	**0.958**	0.028
P TCI	0.072	0.131	**0.893**	-0.046	-0.230	0.114	0.098	-0.054
S TCI	-0.424	**0.721**	0.437	-0.067	0.096	-0.030	-0.003	0.059
C TCI	-0.145	**0.858**	0.047	-0.257	-0.069	0.118	0.243	-0.156
ST TCI	0.160	0.068	0.062	-0.041	-0.091	**0.963**	-0.030	0.081
S SCL	**0.937**	-0.068	0.052	-0.134	-0.009	0.106	-0.040	-0.009
OC SCL	**0.736**	-0.006	-0.291	0.206	-0.282	0.096	-0.137	0.312
IS SCL	**0.709**	-0.239	-0.190	0.475	-0.126	-0.057	-0.129	0.216
D SCL	**0.868**	-0.067	-0.181	0.196	-0.143	-0.010	-0.125	0.144
A SCL	**0.795**	-0.296	-0.038	0.375	-0.001	0.121	-0.007	0.076
AH SCL	0.480	-0.100	-0.035	0.059	-0.034	0.105	0.047	**0.828**
PA SCL	0.422	-0.280	-0.107	**0.818**	-0.009	-0.072	-0.091	0.053
PI SCL	**0.756**	-0.133	0.106	0.275	0.016	0.206	-0.011	0.319
P SCL	**0.875**	-0.255	0.121	0.177	-0.030	0.053	-0.056	0.154

Table A.59: Depressed males TCI and SCL at six months, eight component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7	PC 8	PC 9
NS TCI	-0.046	-0.016	0.014	-0.015	**0.658**	-0.178	-0.011	-0.030	-0.029
HA TCI	0.153	**-0.765**	0.274	0.065	-0.296	0.194	-0.086	-0.007	-0.132
RD TCI	-0.162	0.259	-0.131	0.038	0.088	0.093	-0.054	-0.049	-0.058
P TCI	0.152	**0.770**	0.043	-0.054	-0.490	0.130	-0.062	-0.066	-0.165
S TCI	-0.328	**0.699**	-0.219	-0.061	-0.119	-0.132	-0.004	0.187	-0.017
C TCI	-0.153	**0.401**	-0.399	-0.124	-0.124	0.110	-0.046	0.356	0.041
ST TCI	0.147	-0.029	-0.076	0.091	-0.262	**0.766**	0.030	0.020	-0.005
S SCL	**0.964**	-0.082	0.008	0.113	0.009	0.065	-0.009	0.033	-0.122
OC SCL	**0.594**	-0.441	0.199	0.400	-0.358	0.017	0.148	0.309	-0.008
IS SCL	**0.574**	-0.427	0.461	0.275	-0.233	-0.148	0.219	-0.201	0.179
D SCL	**0.763**	-0.309	0.259	0.246	-0.176	0.010	-0.037	0.086	0.392
A SCL	**0.716**	-0.273	0.508	0.215	-0.063	0.155	-0.172	-0.180	-0.035
AH SCL	0.366	-0.039	0.134	**0.909**	-0.002	0.118	0.042	-0.043	0.022
PA SCL	0.290	-0.288	**0.892**	0.111	0.008	-0.089	0.070	0.014	0.038
PI SCL	**0.668**	-0.063	0.357	0.302	-0.021	0.279	0.493	-0.032	-0.017
P SCL	**0.814**	-0.109	0.312	0.206	-0.091	0.151	0.165	-0.163	0.087
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8	IC 9
NS TCI	0.030	-0.177	-0.051	-0.490	0.092	0.044	-0.172	-0.579	**-1.534**
HA TCI	-0.235	-0.120	-0.058	0.083	0.570	-0.220	**-1.752**	-0.133	1.161
RD TCI	-0.014	0.085	-0.047	-0.029	-0.056	-0.086	0.024	0.117	0.050
P TCI	0.126	0.076	0.221	0.769	0.302	-0.273	**2.058**	1.679	1.857
S TCI	0.138	-0.092	-0.201	0.188	-0.014	0.221	**1.546**	0.567	0.232
C TCI	-0.112	-0.048	-0.272	-0.302	-0.278	0.174	0.493	0.056	0.347
ST TCI	-0.209	0.708	-0.306	-0.840	-0.933	-0.260	-1.465	0.202	**1.980**
S SCL	-0.920	-0.638	1.088	-0.145	**1.513**	-0.328	0.900	-0.385	-1.513
OC SCL	-0.549	-0.634	-0.648	-0.089	**1.565**	1.254	0.712	-0.550	0.581
IS SCL	0.740	0.574	**1.488**	2.354	0.250	0.164	0.017	0.051	0.084
D SCL	-0.122	-0.206	-0.991	-0.565	**-2.555**	-0.479	0.372	-1.034	0.308
A SCL	-0.118	-0.479	-0.110	-0.119	0.765	**-1.760**	-0.621	0.741	**0.645**
AH SCL	-0.232	1.030	**-1.296**	0.600	0.242	-0.525	-0.036	0.434	-0.773
PA SCL	1.364	-0.835	-1.231	**-1.434**	0.283	0.335	0.996	0.230	-0.535
PI SCL	0.414	1.421	0.669	**-1.796**	-0.514	1.079	-0.574	-0.299	0.062
P SCL	0.021	0.515	**0.724**	-0.112	-0.328	-0.279	-0.035	0.120	0.226
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8	FA 9
NS TCI	-0.073	-0.119	-0.039	0.000	**0.677**	-0.027	-0.117	0.074	-0.012
HA TCI	0.242	**-0.657**	-0.250	0.325	-0.539	-0.018	0.207	-0.013	-0.091
RD TCI	-0.132	0.091	0.168	-0.070	0.068	0.022	-0.029	**0.543**	-0.002
P TCI	0.059	**0.756**	0.049	-0.035	-0.302	-0.060	0.148	0.146	-0.010
S TCI	-0.427	**0.543**	0.519	-0.122	0.074	0.033	-0.013	0.062	-0.026
C TCI	-0.166	0.141	**0.869**	-0.213	-0.067	-0.124	0.141	0.335	-0.024
ST TCI	0.168	0.048	0.075	-0.041	-0.149	0.085	**0.594**	-0.025	0.025
S SCL	**0.853**	0.010	-0.065	-0.034	-0.043	0.031	0.259	-0.055	0.000
OC SCL	**0.669**	-0.281	0.013	0.179	-0.289	0.366	0.148	-0.225	0.069
IS SCL	**0.701**	-0.226	-0.196	0.421	-0.143	0.244	-0.087	-0.217	0.107
D SCL	**0.869**	-0.177	-0.007	0.159	-0.153	0.204	-0.054	-0.233	-0.054
A SCL	**0.793**	-0.081	-0.265	0.448	0.021	0.110	0.197	-0.029	-0.206
AH SCL	0.478	-0.023	-0.113	0.089	-0.018	**0.695**	0.166	0.061	0.043
PA SCL	0.419	-0.171	-0.244	**0.746**	-0.038	0.085	-0.108	-0.156	0.076
PI SCL	**0.719**	0.035	-0.135	0.283	-0.007	0.247	0.291	-0.016	0.488
P SCL	**0.869**	0.049	-0.247	0.181	-0.078	0.121	0.099	-0.054	0.139

Table A.60: Depressed males TCI and SCL at six months, nine component solution.

Variables	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7	PC 8	PC 9	PC 10	PC 11	PC 12
NS TCI	-0.046	0.113	0.012	**0.816**	-0.030	-0.020	-0.105	-0.070	-0.008	0.044	-0.033	-0.041
HA TCI	0.182	**-0.826**	0.274	-0.324	-0.063	0.037	0.120	0.191	-0.068	-0.001	0.042	-0.080
RD TCI	-0.110	0.080	-0.072	-0.010	**0.759**	0.031	-0.103	-0.095	0.021	0.051	-0.006	-0.031
P TCI	0.130	**0.648**	0.006	-0.613	0.067	-0.017	0.094	-0.134	0.009	0.221	-0.125	-0.170
S TCI	-0.353	**0.761**	-0.136	-0.032	0.275	-0.073	0.051	0.066	-0.112	-0.115	0.019	-0.057
C TCI	-0.151	0.382	-0.280	-0.109	**0.606**	-0.163	0.163	0.181	-0.116	-0.250	-0.075	0.066
ST TCI	0.134	0.000	-0.050	-0.142	-0.060	0.081	**0.953**	0.029	0.036	0.000	-0.006	0.000
S SCL	**0.954**	-0.071	-0.007	-0.045	-0.077	0.104	0.104	0.030	-0.020	-0.147	-0.069	-0.162
OC SCL	0.589	-0.278	0.186	-0.152	-0.161	0.308	0.090	**0.614**	0.075	-0.019	0.067	0.046
IS SCL	**0.601**	-0.297	0.444	-0.068	-0.224	0.228	-0.047	0.173	0.124	0.029	0.438	0.083
D SCL	**0.787**	-0.212	0.241	-0.096	-0.145	0.203	0.036	0.206	-0.064	-0.038	0.130	0.375
A SCL	**0.752**	-0.257	0.457	0.037	-0.068	0.194	0.135	0.031	-0.090	0.286	0.056	-0.034
AH SCL	0.385	-0.061	0.115	-0.016	-0.008	**0.900**	0.099	0.089	0.067	0.020	0.031	0.017
PA SCL	0.320	-0.249	**0.870**	0.015	-0.213	0.101	-0.088	0.066	0.089	0.006	0.025	0.027
PI SCL	**0.681**	-0.064	0.308	-0.029	-0.015	0.283	0.205	0.098	0.543	0.001	0.062	-0.030
P SCL	**0.828**	-0.120	0.207	-0.105	-0.193	0.204	0.019	0.002	0.271	0.234	0.032	0.088
Variables	IC 1	IC 2	IC 3	IC 4	IC 5	IC 6	IC 7	IC 8	IC 9	IC 10	IC 11	IC 12
NS TCI	-0.104	0.971	-0.283	0.066	-0.021	0.895	0.581	0.089	0.171	**-2.326**	-1.062	0.273
HA TCI	0.646	-1.993	0.184	-0.467	0.168	-0.017	0.355	-0.475	0.108	1.738	**2.434**	-0.990
RD TCI	1.099	1.203	-0.163	-0.288	-0.872	1.036	-0.736	**-2.303**	-1.048	0.838	2.267	-1.497
P TCI	-0.210	0.590	0.400	0.185	-0.006	-1.732	-0.770	-0.134	0.519	**2.623**	-1.087	0.322
S TCI	0.455	1.166	-0.573	0.355	-0.863	0.933	**-1.420**	-0.540	0.297	0.334	-1.087	0.361
C TCI	1.029	0.847	-0.585	-0.129	-0.757	**1.686**	-1.416	-1.541	-0.641	0.818	1.166	-0.632
ST TCI	-1.281	-2.019	-0.604	0.231	0.125	**2.686**	0.464	2.029	0.559	0.994	-2.310	1.194
S SCL	0.916	-2.082	0.889	-0.644	0.375	-0.401	**-2.295**	-0.481	1.612	-1.130	-0.456	0.236
OC SCL	0.224	**1.948**	-0.676	-0.609	0.308	-0.277	0.688	0.274	1.665	-0.294	0.243	1.158
IS SCL	0.042	-0.450	1.259	0.954	**-2.730**	0.261	-0.495	-0.018	0.619	0.048	-0.414	0.426
D SCL	0.043	-0.433	-0.966	-0.147	0.652	0.208	-0.420	0.195	**-2.684**	0.325	0.296	0.839
A SCL	0.561	1.155	-0.252	-0.494	-0.477	1.135	1.848	**-1.897**	0.163	0.380	-1.055	-0.684
AH SCL	-0.942	-0.979	**-1.186**	-0.111	-0.445	-0.866	-0.444	-0.294	0.128	-0.185	-0.066	-0.764
PA SCL	0.684	-0.629	-1.209	1.499	**1.921**	-0.564	-1.232	0.373	0.459	-0.121	-0.319	-0.014
PI SCL	-1.024	0.738	0.645	0.489	0.852	**1.757**	-0.550	-0.076	-0.548	0.013	1.641	-0.754
P SCL	-1.332	0.908	1.262	-0.267	1.150	**-2.082**	1.842	0.460	-0.703	0.096	0.105	0.036
Variables	FA 1	FA 2	FA 3	FA 4	FA 5	FA 6	FA 7	FA 8	FA 9	FA 10		
NS TCI	-0.070	-0.052	-0.001	-0.039	-0.084	**0.732**	-0.030	0.061	-0.011	-0.017		
HA TCI	0.243	**-0.739**	0.301	-0.173	0.124	-0.458	0.025	-0.053	-0.050	-0.207		
RD TCI	-0.131	0.092	-0.082	0.175	-0.026	0.051	0.018	**0.495**	-0.002	-0.003		
P TCI	0.093	**0.691**	-0.032	0.038	0.110	-0.364	-0.061	0.158	-0.003	-0.104		
S TCI	-0.410	**0.596**	-0.123	0.493	0.000	0.026	0.014	0.059	-0.043	-0.021		
C TCI	-0.145	0.173	-0.221	**0.868**	0.095	-0.077	-0.112	0.344	-0.027	0.007		
ST TCI	0.149	0.028	-0.038	0.062	**0.973**	-0.130	0.078	-0.032	0.031	-0.005		
S SCL	**0.912**	-0.005	-0.055	-0.025	0.105	-0.035	0.052	-0.103	0.026	-0.168		
OC SCL	**0.653**	-0.307	0.192	0.049	0.077	-0.246	0.402	-0.266	0.075	0.001		
IS SCL	**0.649**	-0.255	0.455	-0.199	-0.042	-0.116	0.252	-0.233	0.094	0.137		
D SCL	**0.832**	-0.205	0.215	-0.021	0.022	-0.131	0.205	-0.234	-0.094	0.249		
A SCL	**0.785**	-0.125	0.447	-0.252	0.125	0.007	0.142	-0.017	-0.126	-0.093		
AH SCL	0.470	-0.027	0.096	-0.111	0.107	-0.019	**0.712**	0.053	0.057	0.006		
PA SCL	0.381	-0.198	**0.766**	-0.235	-0.079	-0.004	0.088	-0.173	0.083	0.004		
PI SCL	**0.722**	0.012	0.275	-0.120	0.180	-0.038	0.275	-0.015	0.529	-0.007		
P SCL	**0.854**	0.010	0.216	-0.247	0.063	-0.095	0.148	-0.050	0.146	0.052		

Table A.61: Depressed males TCI and SCL at six months, twelve component solution.

Comparison Across Time

At baseline, one, three, five, six and seven components were retained. After treatment two, three, five, eight, nine and twelve components were retained. The three component solutions are presented in Tables A.51 and A.57. The solutions are different after treatment compared to baseline. Likewise the five component solutions are very different at the two time points (Tables A.52 and A.58).

A.10 Comparison of the Personality and Symptoms of the Males and Females

At Baseline

The females, at baseline, retained two, three, four, seven and ten components; whereas the males, at baseline, retained one, three, five, six and seven components. Comparison of the three component solutions, from Tables A.38 and A.51, shows that quite different structures were obtained. Interestingly the personality variables that were in the PC models were self directedness and cooperativeness for both the males and females. In both cases the IC model had one redundant component and two components contrasting the symptom variables.

The first component of the factor model is similar across males and females. Both are a weighted average of a subset of symptom variables. The only difference in the first component is that the female's component also includes self transcendence. The remaining two components are quite different when comparing the males to the females.

The seven component models can also be compared. These models are presented in Tables A.40 and A.47. Three of the seven PC components are similar across gender. The IC solution has two components that are similar and both have two redundant IC components. The FA components are different across gender.

After Treatment

One, four, five, six, seven and eight components were retained for the female models after treatment; in comparison two, three, five, eight, nine and twelve components were retained for the males after treatment. The five component solutions can be compared and the components are presented in Tables A.45 and A.58. The PC and IC solutions are very different across gender. However, the first component in the FA model, which measures a weighted average of all the symptom variables, is similar for the males and females. The highest loading symptom is anxiety for the females and psychotocism for the males. So the first component has the same variables for males and females but there are

differences in the relative loadings across the variables. The male's third FA component is similar to the female's fourth FA component. The second FA component for the males appears to be the combined version of the second and third female components. The two components of the male's FA solution are redundant and the last female component is redundant.

The eight component solutions can also be compared (Tables A.48 and A.59). The PC solution has one common component across males and females. This component is essentially anger hostility. The IC solution has one common component across males and females, a single indicator of anxiety. The FA solution has three components that are similar between males and females.

A.11 Summary

This appendix has presented the models that were developed in Chapter 3. The personality models showed differences both across time and gender. The symptom models at baseline were calculated on the combined data from the males and females, as the Flury test had indicated that there was a common structure across gender. After treatment however there were significant differences between the males and females so the symptom models were developed separately. Comparison of those after treatment models showed in general that the components were in fact different after treatment. Likewise the combined symptom and personality models had few components that were similar across males and females.

In general the comparisons across time showed few similarities between components. The exception to this was the depressed male's symptom structures. There were a number of components that were similar across time. The female's personality structures after treatment were generally different to those at baseline. The same trend was seen with the male's personality across time. The female's symptom structures also appeared different after treatment. Similar results were seen in the combined symptoms and personality models.

As far as the author is aware, this is the first study to combine all of the TCI and SCL variables into a components analysis to investigate the covariance structure not only within symptoms and personality but across them as well. In general the PC and IC models had little mixing of the personality and symptom variables. The FA models had more mixing across symptoms and personality. The symptom variables tended to dominate, particularly in the models with a small number of retained components.

The PC, IC and FA models were often quite different. It is clear that each method is investigating different aspects of the data. The PC models are aiming for decorrelated or orthogonal components. The IC method extends this to aim for independent components.

Neither of these methods allowed for a noise or error term for each variable. The factor analysis method specifically finds components that model the common variance across the variables rather than the specific variance. The methods gave the most similar results when only one component was retained.

It is interesting to note that all the PC and FA symptom models have positive loadings suggesting that the symptoms are measured in a "similar direction", however the IC models, in some cases, have contrasting loadings. This suggests that to obtain independence rather than just decorrelated constructs the symptoms are not all measured in a similar direction.

The IC models tended to be harder to interpret. The models were not scaled to have loadings between zero and one. Sometimes the loadings were very small across the board and at other times the loadings were large across the board (compared to the PC and FA solutions). This meant a judgement call had to made for each model as to how high a loading was before it was included in the model. Generally this problem was bypassed by highlighting the highest loading for the variable (by nature of the type of models needed for the confirmatory factor analysis), however if all the loadings were small a judgement call had to be made whether the highest loading was large enough to be included or not. The IC solutions tended to be more likely to have a variable with large loadings on more than one component. This is one area in which future work can be done, the confirmatory models could be allowed to be more complex and allow variables to load on more than one factor. This should benefit the IC type models where this appears to occur more often. However in this study, due to sample size restricting the complexity of the models, the standard confirmatory factor analysis model will be used.

The models developed in this chapter will be cross validated using confirmatory factor analysis in Chapter 4. At present there is no way to distinguish between the models presented here hence the use of the confirmatory factor analysis on a second dataset to find the best model.

Appendix B

Combining Personality and Symptoms in Confirmatory Factor Analysis Models

Component analysis was performed on the combined personality and symptom variables to investigate the covariance structure between them. In the confirmatory analyses these models were compared to the combination of the best individual TCI and SCL models from the previous section, when available, that were then combined in one model and allowed to covary across personality and symptoms. Tables B.1 to B.10 present the combined TCI and SCL results for the males and females at both time points.

The Females at Baseline

At baseline the females 7 IC and 10 IC models fit the data reasonably well (Table B.1). The 10 component IC solution appears to have a slightly better fit. Both will be retained for bootstrapping and model comparison.

Figure B.1 presents the seven component IC model and Figure B.2 presents the ten component IC model. Due to redundant factors, these are factors that no variable loads highest on, the seven component model actually has five factors. These five factors are made up entirely from the symptom variables. The loadings are all non zero and positive. The error variances, likewise are all positive.

The Bollen-Stine transformation (Bollen and Stine, 1992) could not be performed on this model due to problems with the Cholesky factorisation of the estimated covariance matrix. This problem was unable to be resolved. The naïve bootstrapped fit indices are also low suggesting a poor fit and it is unclear whether this would be resolved by bootstrapping with the transformation (Bollen and Stine, 1992) as naïve bootstrapping tends be overly conservative in estimating the fit indices.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
2 PCs	0.849	0.755	**0.057**	0.126	0.916	31.195	0.880
3 PCs	0.841	0.744	**0.062**	0.125	0.905	35.808	0.863
4 PCs	0.866	0.762	**0.055**	0.130	0.919	31.919	0.886
7 PCs	0.874	0.771	**0.067**	0.114	0.922	23.785	0.881
10 PCs	0.864	0.714	**0.079**	0.116	0.900	34.622	0.855
2 ICs	0.887	0.790	**0.056**	0.134	0.930	17.358	**0.902**
3 ICs	0.882	0.775	**0.052**	0.132	0.934	20.924	**0.907**
4 ICs	0.877	0.739	**0.050**	0.154	0.921	31.117	0.897
7 ICs	**0.910**	0.799	**0.046**	**0.075**	**0.956**	10.514	**0.933**
10 ICs	**0.925**	0.823	**0.059**	**0.079**	**0.967**	**-8.137**	**0.929**
2 FAs	0.792	0.712	0.0848	0.119	0.857	53.711	0.794
3 FAs	0.770	0.682	0.0911	0.126	0.826	78.243	0.760
4 FAs	0.785	0.692	0.0892	0.126	0.830	77.012	0.767
7 FAs	0.787	0.660	0.0856	0.119	0.852	58.490	0.790
10 FAs	0.855	0.722	**0.074**	0.104	0.905	20.848	0.851
TCI 1 SCL 6	0.884	0.7650	**0.059**	0.105	0.928	14.820	0.884

Table B.1: The female's combined symptom and personality models at baseline using the confirmatory dataset.

Covariance	Median	(90% CI)
Factor 1 Factor 2	0.668	(0.584, 0.738)
Factor 1 Factor 3	0.719	(0.616, 0.813)
Factor 2 Factor 3	1.079	(1.031, 1.144)
Factor 1 Factor 4	0.608	(0.533, 0.671)
Factor 2 Factor 4	0.958	(0.937, 0.980)
Factor 4 Factor 4	0.868	(0.808, 0.933)
Factor 1 Factor 6	0.508	(0.395, 0.609)
Factor 2 Factor 6	0.869	(0.812, 0.910)
Factor 3 Factor 6	0.901	(0.839, 0.956)
Factor 4 Factor 6	0.689	(0.625, 0.746)

Table B.2: The bootstrapped parameter estimates for the females baseline TCI and SCL 7 IC model.

	Naïve Bootstrap	
Fit Index	Median	(90% CI)
GFI	0.834	(0.790, 0.876)
AGFI	0.627	(0.528, 0.722)
RMR	0.303	(0.244, 0.372)
Chi-Square	159.051	(116.077, 208.639)
df	16	
p-value	0.000	(0.000, 0.000)
RMSEA	0.201	(0.168, 0.233)
CFI	0.853	(0.790, 0.903)
AIC	127.051	(84.077, 176.639)
NFI	0.841	(0.779 , 0.890)

Table B.3: The bootstrapped fit indices for the females baseline TCI and SCL 7 IC model.

Table B.2 presents the covariance estimates with their 90 % confidence intervals. The intervals are all non zero and positive suggesting that all the covariances are significant and positive. This is the same pattern seen in the symptom models presented previously (Table 4.9, Figure 4.5). The symptoms load positively and the factors are positively interrelated reinforcing the idea of the symptoms all working the same direction.

The ten component IC solution has one redundant factor (Figure B.2), in fact nine components are shown in the model. This combined model does, however, have some contributions from the personality variables. Novelty seeking and reward dependence load on the same factor as interpersonal sensitivity (Factor 5), but the TCI traits have small loadings and large error variances, making them poor predictors of Factor 5. Personality is again involved in Factor 8. Persistence is a single indicator for this factor. Interestingly Factor 5 and Factor 8 do not have any significant covariances with any of the other factors. Thus the factors containing personality variables do not have any significant covariances with the other symptoms indicating that personality and symptoms are distinct with little covariance between them. There is only one factor that has both symptom and personality variables.

The Females After Treatment

Table B.6 presents the results of the females TCI and SCL combined models post treatment. Clearly all the models are a poor fit. It would be unlikely for the best of these to perform better with bootstrapping as the number of parameters in the model is fairly large compared to the sample size.

Covariance	Median	(90% CI)	Covariance	Median	(90% CI)
Factor 1 Factor 2	0.628	(-0.004, 0.711)	Factor 1 Factor 3	0.517	(-0.162, 0.635)
Factor 2 Factor 3	0.736	(0.160, 0.790)	Factor 1 Factor 5	-0.285	(-1.936, 0.847)
Factor 2 Factor 5	-0.684	(-2.606, 1.029)	Factor 3 Factor 5	-0.655	(-2.366, 0.990)
Factor 1 Factor 6	0.613	(0.045, 0.721)	Factor 2 Factor 6	0.738	(0.514, 0.803)
Factor 3 Factor 6	0.769	(0.383, 0.841)	Factor 5 Factor 6	-0.575	(-2.451, 1.039)
Factor 1 Factor 7	0.369	(-0.328, 0.672)	Factor 2 Factor 7	0.233	(-0.362, 0.753)
Factor 3 Factor 7	0.445	(-0.347, 0.818)	Factor 5 Factor 7	0.275	(-1.572, 0.947)
Factor 6 Factor 7	0.537	(-0.176, 0.920)	Factor 1 Factor 8	-0.426	(-0.646, 0.598)
Factor 2 Factor 8	-0.560	(-0.685, 0.645)	Factor 3 Factor 8	-0.548	(-0.737, 0.669)
Factor 5 Factor 8	0.617	(-0.972, 2.046)	Factor 6 Factor 8	-0.368	(-0.767, 0.774)
Factor 7 Factor 8	0.667	(-0.787, 0.801)	Factor 1 Factor 4	0.654	(-0.217, 0.739)
Factor 2 Factor 4	0.779	(-0.281, 0.825)	Factor 3 Factor 4	0.748	(-0.346, 0.824)
Factor 4 Factor 5	-0.037	(-2.638, 1.056)	Factor 4 Factor 6	0.821	(-0.027, 0.929)
Factor 4 Factor 7	0.835	(-0.406, 0.890)	Factor 4 Factor 8	-0.740	(-0.826, 0.794)
Factor 1 Factor 9	0.576	(-0.093, 0.666)	Factor 2 Factor 9	0.644	(-0.009, 0.716)
Factor 3 Factor 9	0.717	(-0.032, 0.782)	Factor 5 Factor 9	-0.416	(-2.324, 0.933)
Factor 6 Factor 9	0.864	(0.459, 0.916)	Factor 7 Factor 9	0.607	(-0.238, 0.863)
Factor 8 Factor 9	-0.580	(-0.726, 0.642)	Factor 4 Factor 9	0.792	(-0.055, 0.838)

Table B.4: The bootstrapped parameter estimates for the females baseline TCI and SCL 10 IC model.

	Naïve Bootstrap		Bollen-Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.844	(0.794, 0.867)	0.976	(0.963, 0.985)
AGFI	0.621	(0.499, 0.675)	0.940	(0.910, 0.963)
RMR	0.534	(0.345, 138.577)	1.818	(0.282, 5.082)
Chi-Square	608.104	(540.351, 1842.600)	33.984	(20.779, 52.429)
df	32		32	
p-value	0.000	(0.000, 0.000)	0.372	(0.936, 0.013)
RMSEA	0.285	(0.268, 0.506)	0.017	(0.000, 0.054)
CFI	0.531	(-0.457, 0.618)	0.999	(0.993, 1.000)
AIC	544.150	(476.351, 1778.360)	-30.019	(-43.238, -11.571)
NFI	0.530	(-0.406, 0.613)	0.989	(0.983, 0.993)

Table B.5: The bootstrapped fit indices for the females baseline TCI and SCL 10 IC model.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC/FA	0.718	0.577	**0.078**	0.1928	0.8276	63.9315	0.7770
4 PCs	0.748	0.590	**0.074**	0.1662	0.8618	39.4878	0.8082
5 PCs	0.738	0.582	**0.076**	0.1611	0.8525	40.6305	0.7910
6 PCs	0.778	0.639	**0.073**	0.1472	0.8660	**24.1251**	0.7986
7 PCs	-	-	-	-	-	-	-
8 PCs	0.779	0.611	0.0885	0.154	0.847	37.690	0.782
1 IC	0.701	0.502	0.0841	0.239	0.822	74.518	0.788
4 ICs	0.732	0.606	0.0985	0.170	0.819	48.083	0.753
5 ICs	0.717	0.595	0.0907	0.157	0.800	51.815	0.719
6 ICs	0.730	0.590	0.0900	0.152	0.823	42.084	0.746
7 ICs	0.732	0.599	0.0907	0.147	0.816	39.327	0.733
8 ICs	0.752	0.594	0.1890	0.146	0.835	33.943	0.759
4 FAs	0.704	0.577	0.0851	0.164	0.788	64.756	0.712
5 FAs	0.701	0.583	0.0873	0.163	0.786	64.691	0.707
6 FAs	0.697	0.556	0.1688	0.159	0.783	61.189	0.702
7 FAs	0.731	0.588	0.0815	0.151	0.810	45.597	0.730
8 FAs	0.709	0.556	0.1655	0.164	0.778	68.605	0.703
TCI 1 SCL 2	0.551	0.3110	0.363	0.333	0.325	272.273	0.314

Table B.6: The female's combined symptom and personality models after treatment using the confirmatory dataset.

	Fit Indices						
Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
1 PC	0.706	0.559	0.103	0.160	0.794	6.492	0.686
3 PCs	0.732	0.568	0.109	0.159	0.810	5.736	0.708
5 PCs	0.719	0.536	0.101	0.165	0.800	9.006	0.704
6 PCs	0.861	0.706	**0.070**	0.112	0.938	**-11.458**	0.855
7 PCs	0.715	0.506	0.120	0.178	0.763	19.372	0.676
1 IC	0.715	0.525	0.097	0.185	0.821	14.340	0.745
3 ICs	0.716	0.509	0.097	0.190	0.818	16.147	0.745
5 ICs	0.879	0.727	**0.083**	0.130	0.934	-3.779	0.867
6 ICs	0.873	0.696	**0.079**	0.136	0.933	-2.591	0.871
7 ICs	0.819	0.646	**0.078**	0.123	0.932	-7.236	0.855
1 FA	0.669	0.522	0.120	0.171	0.738	17.026	0.629
3 FAs	0.717	0.584	0.114	0.139	0.815	-8.314	0.685
5 FAs	0.682	0.540	0.152	0.161	0.708	15.558	0.589
6 FAs	0.686	0.530	0.131	0.156	0.734	8.763	0.615

Table B.7: The male's combined symptom and personality models at baseline, using the confirmatory data.

The Males at Baseline

Table B.7 represents the results for the males combined personality and symptoms at baseline. The males 6 PC solution appears to fit the best but the indices aren't quite in the correct bounds. This model will be retained as the normality assumptions may be violated, causing poorer model fit at this stage.

Figure B.3 represents the male model at baseline. Cooperativeness is the only TCI variable in the model and has a small loading and large error variance. It is contrasted against anger hostility. The sixth error variance has a confidence interval that includes zero so it is not significantly different from zero. The loadings are all significantly different from zero. Anger hostility has a negative loading but also has negative covariances (Table B.8) with the other factors so once again the symptoms are all loading in the same direction. However cooperativeness is working in opposition to the symptoms. In other words the model suggests that a person who has high symptoms will have low cooperativeness.

Table B.9 presents the bootstrap results for the depressed males baseline TCI and SCL model. Most of the fit indices are in the appropriate bounds when looking at the transformed results. The chi-square has a large confidence interval suggesting that a larger sample should be used. The RMR estimate is also ambiguous suggesting that the

Covariance	Median	(90 % CI)	Covariance	Median	(90 % CI)
Factor 1 Factor 2	0.858	(0.804, 0.900)	Factor 1 Factor 3	-0.567	(-0.724, -0.355)
Factor 2 Factor 3	-0.708	(-0.859, -0.504)	Factor 1 Factor 4	0.709	(0.630, 0.772)
Factor 2 Factor 4	0.846	(0.771, 0.902)	Factor 3 Factor 4	-0.615	(-0.778, -0.395)
Factor 1 Factor 5	0.779	(0.722, 0.828)	Factor 2 Factor 5	0.837	(0.770, 0.886)
Factor 3 Factor 5	-0.603	(-0.740, -0.411)	Factor 4 Factor 5	0.642	(0.518, 0.734)
Factor 1 Factor 6	0.791	(0.710, 0.856)	Factor 2 Factor 6	0.876	(0.795, 0.934)
Factor 3 Factor 6	-0.707	(-0.875, -0.477)	Factor 4 Factor 6	0.813	(0.742, 0.867)
Factor 5 Factor 6	0.732	(0.630, 0.818)			

Table B.8: The bootstrapped parameter estimates for the males baseline TCI and SCL model.

	Naïve Bootstrap		Bollen-Stine Transformed	
Variable	Median	(90 % CI)	Median	(90 % CI)
GFI	0.844	(0.810, 0.877)	0.957	(0.932, 0.975)
AGFI	0.670	(0.597, 0.741)	0.910	(0.857, 0.948)
RMR	0.310	(0.243, 0.383)	0.056	(0.022, 0.131)
Chi-Square	120.363	(91.269, 154.133)	28.370	(15.696, 47.061)
df	26		26	
p-value	0.000	(0.000, 0.000)	0.340	(0.943, 0.007)
RMSEA	0.172	(0.143, 0.200)	0.027	(0.000, 0.081)
CFI	0.859	(0.792, 0.909)	0.998	(0.980, 1.000)
AIC	68.363	(39.269, 102.133)	-23.630	(-36.304, -4.939)
NFI	0.831	(0.765, 0.881)	0.973	(0.956, 0.985)

Table B.9: The bootstrapped fit indices for the males baseline TCI and SCL model.

Confirmatory Data - Mixed TCI and SCL Models

Model	GFI	AGFI	RMR	RMSEA	CFI	AIC	NFI
2 PCs	0.587	0.392	0.148	0.251	0.680	13.980	0.565
3 PCs	0.590	0.396	0.147	0.251	0.681	13.917	0.565
5 PCs	0.628	0.427	0.111	0.217	0.764	-3.439	0.634
8 PCs	0.548	0.330	0.142	0.258	0.609	26.658	0.504
9 PCs	0.502	0.280	0.130	0.259	0.595	28.614	0.489
12 PCs	0.553	0.293	0.131	0.263	0.621	32.891	0.528
2 ICs	0.685	0.493	0.126	0.175	0.827	**-10.937**	0.665
3 ICs	0.546	0.334	0.147	0.272	0.593	29.775	0.493
5 ICs	0.612	0.312	0.146	0.269	0.749	13.878	0.661
8 ICs	0.588	0.357	0.166	0.252	0.645	20.893	0.540
9 ICs	0.612	0.363	0.119	0.224	0.721	0.094	0.601
12 ICs	0.168	-0.347	0.201	0.434	-0.010	232.894	0.076
2 FAs	0.503	0.330	0.157	0.273	0.533	43.233	0.430
3 FAs	0.478	0.298	0.150	0.274	0.517	50.45	0.418
5 FAs	0.469	0.268	0.150	0.275	0.522	44.670	0.425
8 FAs	0.516	0.234	0.116	0.273	0.592	42.050	0.506
9 FAs	0.507	0.239	0.113	0.268	0.596	38.694	0.505

Table B.10: The males combined symptom and personality models after treatment, using the confirmatory data.

residuals are not as well behaved as we would like. The other fit indices suggest that the model is appropriate for the data and explains the underlying structure of the combined personality and symptom variables.

The Males After Treatment

The results from the confirmatory factor analysis on the males after treatment PCA, ICA and FA models are presented in Table B.10. All the models have poor fit. None of the models have fit indices that are close enough to the bounds that bootstrapping would help. For this reason no models were retained for the combined personality and symptoms of the males after treatment.

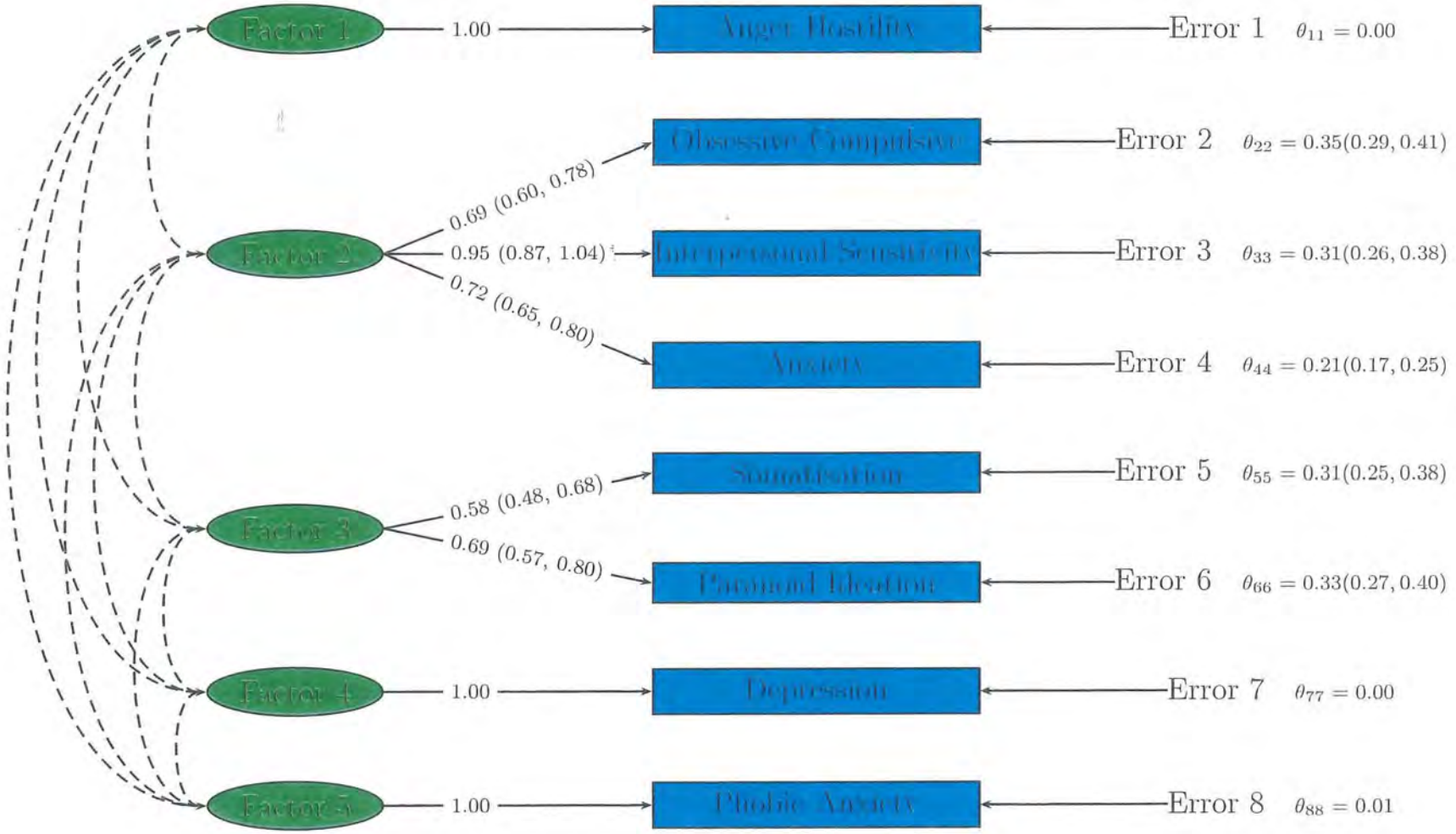


Figure B.1: The depressed females personality and symptom model at baseline (7 IC solution).

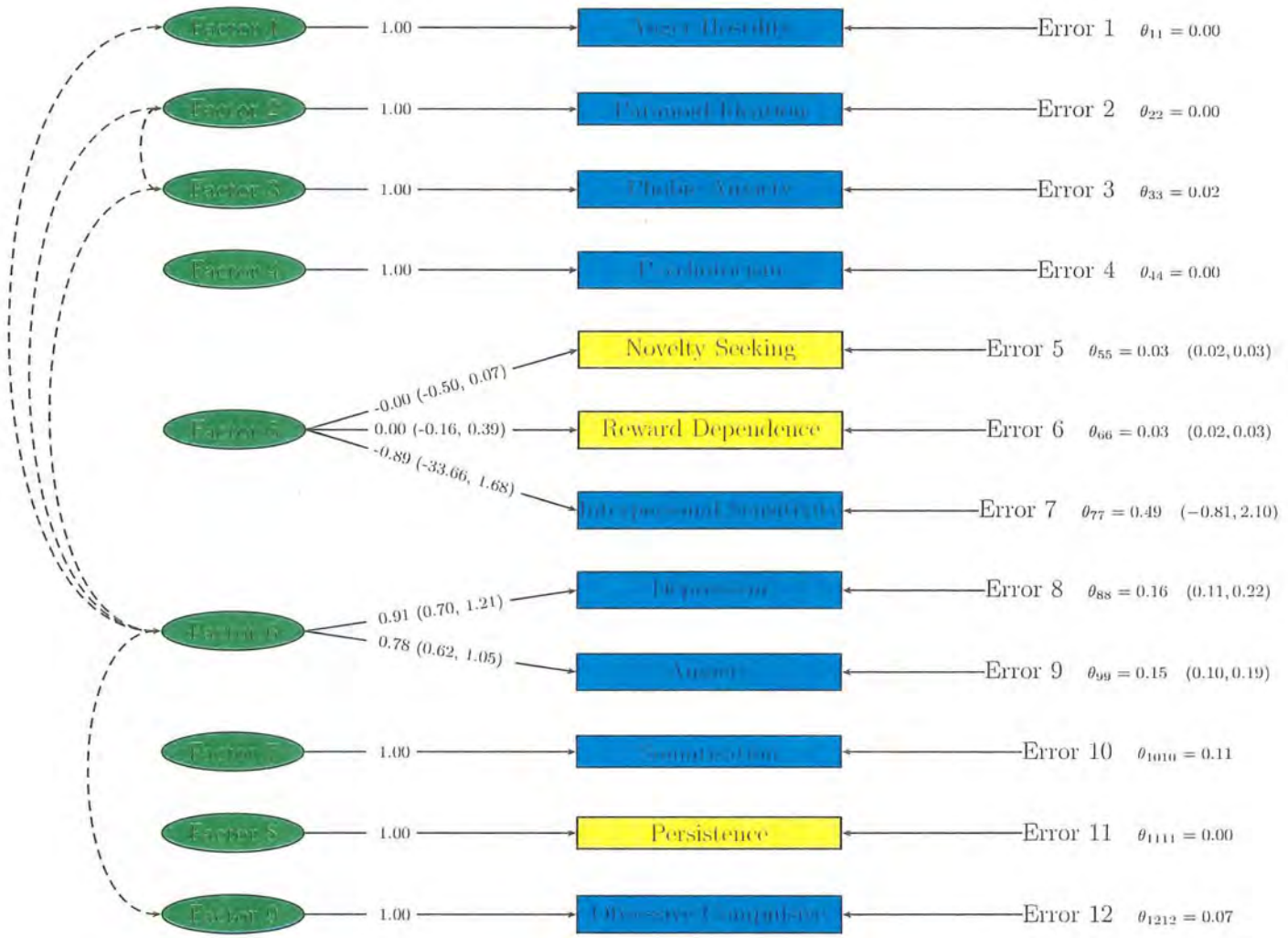


Figure B.2: The depressed females personality and symptom model at baseline (10 IC solution).

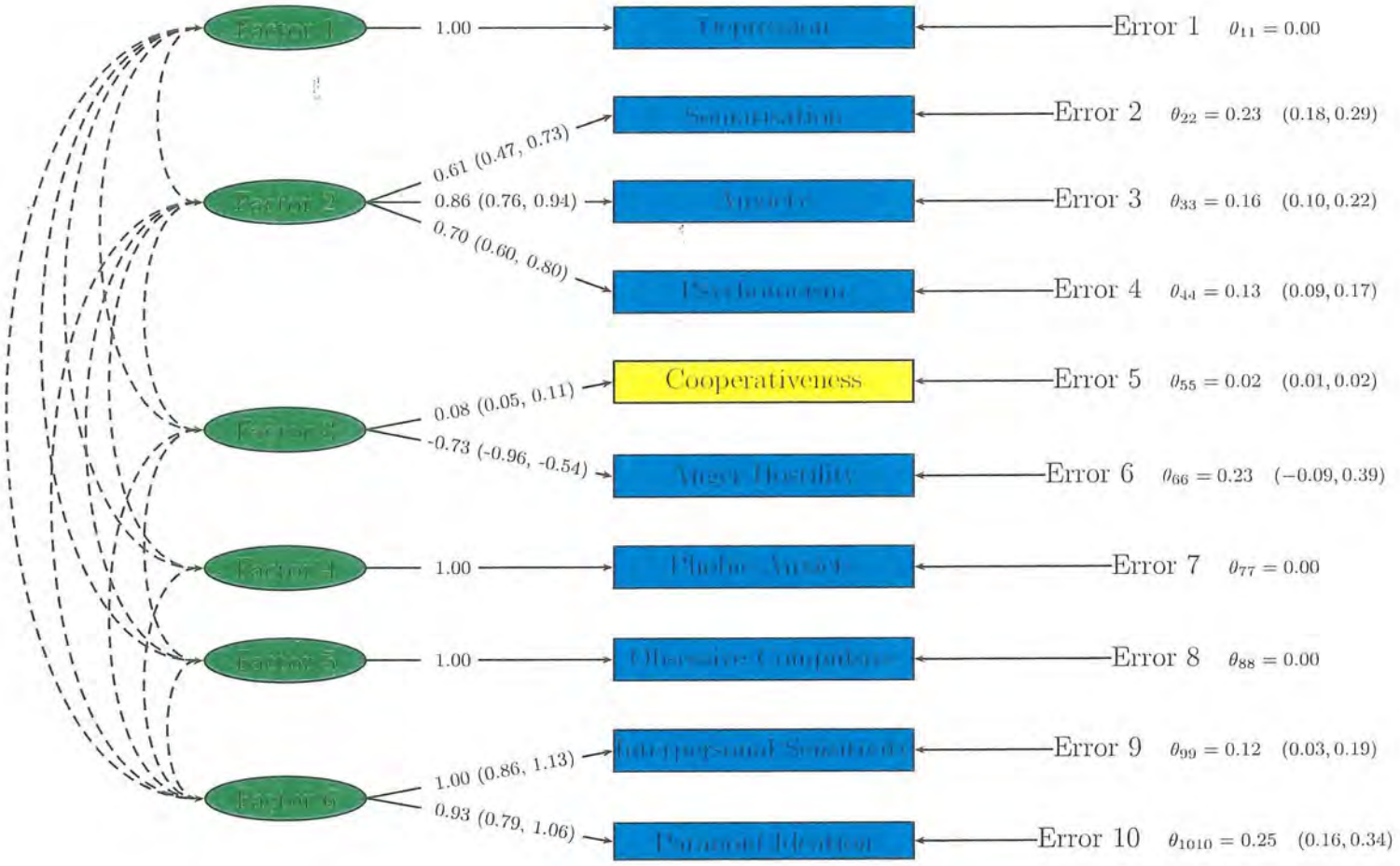


Figure B.3: The depressed males personality and symptom model at baseline.

B.1 Summary of Combined Symptom and Personality Models

At baseline two models were bootstrapped for the females. These two models both described the covariance structure of the combined personality and symptom data. The males had one model bootstrapped at baseline. These models had few contributions from personality. If personality was involved in a latent variable that latent factor tended to have little covariance with the other latent factors. At baseline the symptoms dominate over personality and the two are quite distinct. Therefore it is better to treat symptoms and personality separately, as was done initially, and develop separate models.

After treatment there was no combined symptom and personality model that appeared to fit the data well for either the males or females. All three methods of model development, ICA, PCA and FA, appear to have failed, if we restrict the models to the subset that allows variables to load highly on only one factor.

Appendix C

Predicting Personality from Symptoms of Depression

C.1 Path Analysis Results

The path analysis was conducted and bootstrapped in SAS (SAS(R) Proprietary Software Release (8.1)). Two models were examined, the females at baseline and then the females after treatment.

The Baseline Females

The baseline path diagram is presented in Figure C.1. The left hand side is the structural model for personality and the right hand side is the structural model for symptoms. The two are linked by the arrows going from the latent symptom constructs to the single latent personality construct, which depicts a linear relationship with personality as the dependent variable. The loadings of the personality variables onto the personality factor are not significantly different from zero, as evidenced by the 90% confidence interval containing zero. The regression coefficients from the symptoms to personality are all close to zero and have confidence intervals that contain zero. The bootstrapped fit indices are presented in Table C.1 and indicate that the fit is poor. All the fit indices, both from naïve bootstrapping and using the Bollen-Stine transformation, are outside the appropriate bounds for good fit. The implied covariance structure from the model is significantly different from the sample covariance structure. These results imply that the symptoms are poor predictors of personality for the depressed females at baseline.

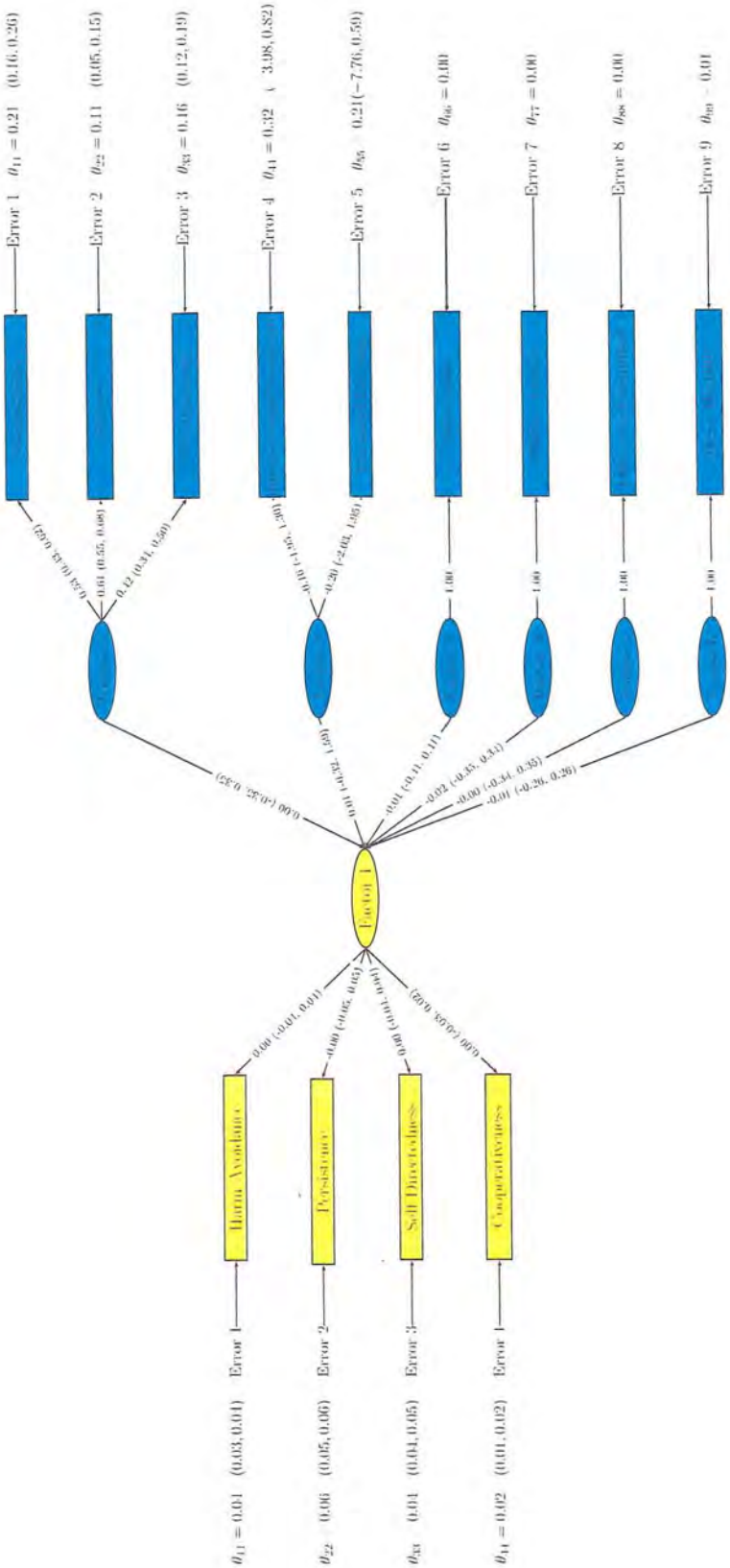


Figure C.1: Depressed females symptoms predicting personality at baseline.

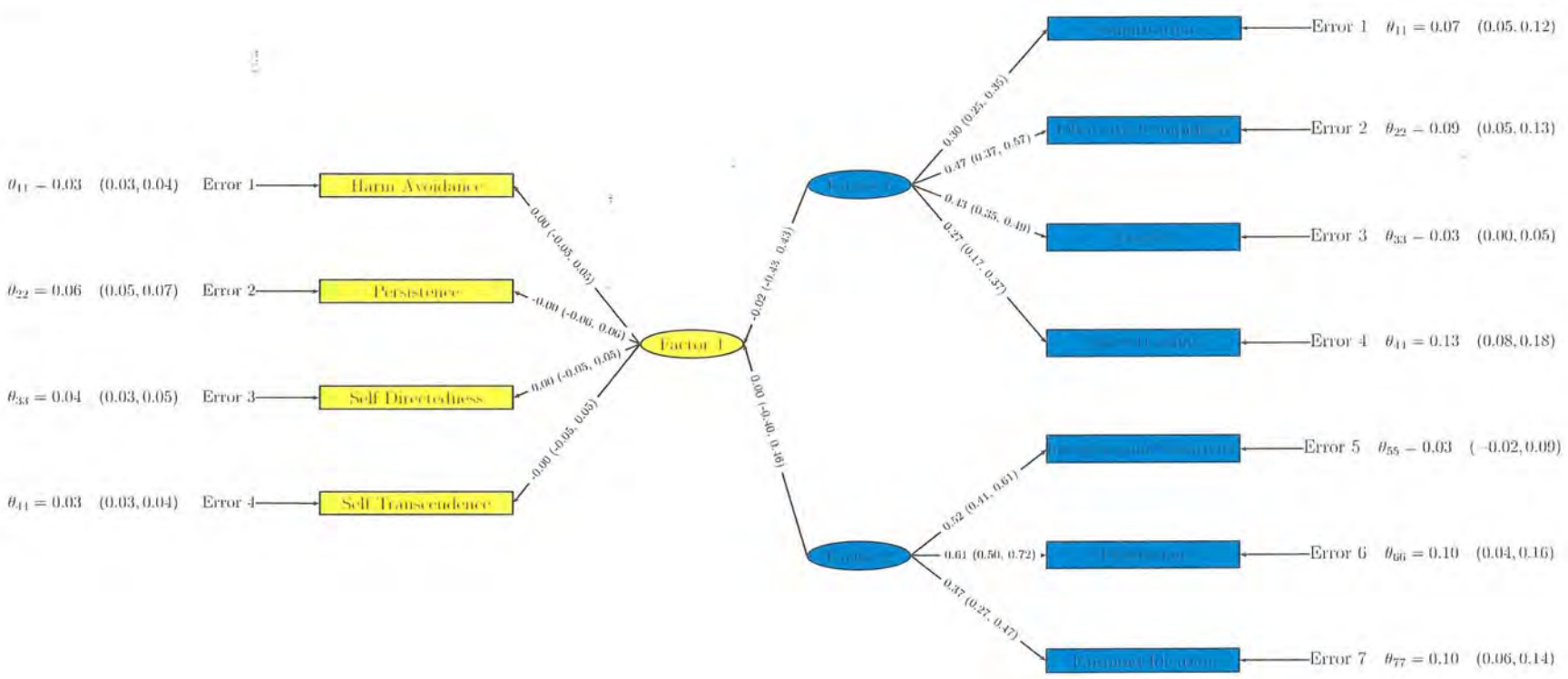


Figure C.2: Depressed females symptoms predicting personality after treatment.

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.489	(0.430, 0.526)	0.685	(0.650, 0.718)
AGFI	0.329	(0.252, 0.378)	0.587	(0.540, 0.630)
RMR	0.187	(0.168, 0.214)	0.096	(0.089, 0.108)
Chi-Square	1297	(1176, 1465)	767	(687, 857)
df	80		80	
p-value	0.000	(0.000, 0.000)	0.000	(0.000, 0.000)
RMSEA	0.262	(0.249, 0.280)	0.197	(0.185, 0.210)
CFI	0.210	(0.108, 0.262)	-0.048	(-0.101, 0.011)
AIC	1137	(1016, 1305)	607	(527, 697)
NFI	0.205	(0.109, 0.255)	-0.028	(-0.072, 0.023)

Table C.1: Fit indices for the baseline female's path analysis model.

The Females After Treatment

Figure C.2 presents the structural model for symptoms as predictors of personality for the depressed females post treatment. The loadings for the personality traits on the underlying personality factor are not significantly different from zero as evidenced by the confidence intervals containing zero. The regression coefficients, from the latent symptom factors to the latent personality factor, are likewise not significantly different from zero. The fit indices are presented in Table C.2 and like the baseline model, most are not in the appropriate bounds, the exception being RMR with a transformed estimate of 0.065. This suggests that post treatment symptoms are poor predictors of personality for the females.

Overview of the Path Analysis

There are a number of possible reasons for the poor model fit. Each model by itself is an adequate representation of the latent structure, from the results of Chapter 4. However when linked together the model fails suggesting that there is not any linear relationship between the latent personality factor and the latent symptom factors. Possible reasons for the poor fit are that latent structures are not important in the relationship or there may in fact be a non-linear relationship between the latent factors. The path analysis agrees with the results found in Appendix B where combined TCI and SCL models were developed. These models had few personality variables in them and, when they were involved in the model, the factors did not have significant covariances with the symptom factors.

	Naïve Bootstrap		Bollen Stine Transformed	
Fit Indices	Median	(90% CI)	Median	(90% CI)
GFI	0.667	(0.631, 0.702)	0.588	(0.549, 0.639)
AGFI	0.510	(0.457, 0.561)	0.394	(0.336, 0.468)
RMR	0.080	(0.059, 0.115)	0.065	(0.049, 0.090)
Chi-Square	491	(428, 566)	585	(478, 690)
df	53		53	
p-value	0.000	(0.000, 0.000)	0.000	(0.000, 0.000)
RMSEA	0.248	(0.230, 0.269)	0.274	(0.245, 0.299)
CFI	0.556	(0.497, 0.609)	0.203	(0.145, 0.350)
AIC	385	(322, 460)	479	(372, 584)
NFI	0.533	(0.477, 0.584)	0.202	(0.150, 0.336)

Table C.2: Fit indices for the post treatment female's path analysis model.

The second method used in this chapter is general additive models (GAMs), which allows for modelling of non-linear relationships. This method is used in the following section both on the latent factors and on the observed variables (TCI traits and SCL symptoms).

C.2 The Best General Additive Models

The Females at Baseline

Table C.3 presents the best GAM models for the depressed females at baseline. The second column presents the dependent personality variable that the model relates to, the third column presents the linear terms in the model and the fourth column presents any non-linear terms in the model. The TCI latent factor has four significant linear latent symptom predictors. Of the seven TCI traits, six TCI traits had significant linear relationships with symptoms and no significant non-linear relationships, the exception was reward dependence.

The Females After Treatment

Table C.4 presents the best GAM models for the females post treatment. The second latent symptom factor (F2 SCL) had a significant non-linear relationship with the latent personality factor. Five of the TCI traits, namely novelty seeking, harm avoidance, reward dependence, persistence and self directedness, had significant non-linear relationships with

Model Number	Personality Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
1	Factor 1 TCI	Factor 1 SCL	
		Factor 2 SCL	
		Factor 3 SCL	
		Factor 4 SCL	
2	Novelty Seeking	Anger Hostility	
3	Harm Avoidance	Interpersonal Sensitivity	
		Paranoid Ideation	
4	Reward Dependence	No Model	
5	Persistence	Somatisation	
		Interpersonal Sensitivity	
6	Self Directedness	Somatisation	
		Interpersonal Sensitivity	
		Psychotocism	
7	Cooperativeness	Anger Hostility	
		Paranoid Ideation	
8	Self Transcendence	Somatisation	
		Obsessive Compulsive	

Table C.3: Best model from GAMs for the depressed females at baseline.

symptoms. Two traits, cooperativeness and self transcendence, had significant linear relationships only, with the symptoms.

Comparison of the Female Models Before and After Treatment

At baseline the TCI factor is modelled by the first four symptom factors, after treatment it is only modelled by the second symptom factor. Remember that at baseline there are six symptom factors and after treatment there are only two symptom factors. At baseline factor one measures somatisation, anxiety and psychotocism, factor two measures interpersonal sensitivity and paranoid ideation, factor three measures depression and factor four measures phobic anxiety. After treatment factor two measures interpersonal sensitivity, depression and paranoid ideation. The post treatment second factor is equivalent to factor two and factor three at baseline.

The personality factor at baseline is a measure of harm avoidance versus persistence, self directedness and cooperativeness. The only difference after treatment is that cooperativeness is replaced by self transcendence in the factor. This suggests that interpersonal sensitivity, depression and paranoid ideation are important in relation to the TCI traits of harm avoidance, persistence and self directedness at both time points.

At baseline harm avoidance is significantly related to interpersonal sensitivity and paranoid ideation. After treatment these symptoms are still significantly related to harm

Model Number	Personality Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
1	Factor 1 TCI		Factor 2 SCL
2	Novelty Seeking	Anxiety	Somatisation
		Paranoid Ideation	
3	Harm Avoidance	Interpersonal Sensitivity	Obsessive Compulsive
		Depression	
		Anger Hostility	
		Paranoid Ideation	
4	Reward Dependence	Paranoid Ideation	Somatisation
5	Persistence	Somatisation	Paranoid Ideation
			Phobic Anxiety
6	Self Directedness	Interpersonal Sensitivity	Psychotocism
7	Cooperativeness	Paranoid Ideation	
		Phobic Anxiety	
8	Self Transcendence	Paranoid Ideation	

Table C.4: Best model from GAMs for the depressed females post treatment.

avoidance. Anger hostility, obsessive compulsive symptoms and depression relate significantly to harm avoidance after treatment. Somatisation is significantly related to persistence both before and after treatment. At baseline interpersonal sensitivity is also related to persistence, whereas after treatment paranoid ideation and phobic anxiety are significantly related to persistence.

Self directedness is significantly related to interpersonal sensitivity and psychotocism at both time points. Somatisation is also significantly related to self directedness, but only at baseline. Cooperativeness relates to anger hostility and paranoid ideation at baseline. After treatment anger hostility is replaced by phobic anxiety in the model.

Novelty seeking, reward dependence and self transcendence have quite different relationships with symptoms before and after treatment. At baseline novelty seeking is related to anger hostility and after treatment it is related to somatisation, anxiety and paranoid ideation. Reward dependence has no significant relationships with the symptoms at baseline, however reward dependence is significantly related to somatisation and paranoid ideation after treatment. Self transcendence relates to somatisation and obsessive compulsive symptoms at baseline and relates to paranoid ideation after treatment.

The only model that depression plays a significant role in for the females is after treatment model number three (Table C.6) which shows that harm avoidance is significantly related to interpersonal sensitivity, depression, anger hostility and obsessive compulsive symptoms.

Model Number	Personality Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
1	Novelty Seeking	No Model	
2	Harm Avoidance	Interpersonal Sensitivity	
3	Reward Dependence	Somatisation	
		Anxiety	
4	Persistence	Obsessive Compulsive	
5	Self Directedness	Obsessive Compulsive	
		Interpersonal Sensitivity	
6	Cooperativeness	Anxiety	
		Anger Hostility	
		Psychotocism	
7	Self Transcendence	Paranoid Ideation	Interpersonal Sensitivity
			Phobic Anxiety

Table C.5: Best model from GAMs for the depressed males at baseline.

The Males at Baseline

The best GAM models for the depressed males at baseline are presented in Table C.5. Harm avoidance, reward dependence, persistence, self directedness and cooperativeness had linear relationships with some of the symptom variables. Self transcendence had both linear and non-linear relationships with three of the symptom variables. Novelty seeking had no significant relationships.

The Males After Treatment

Table C.6 presents the best models for the post treatment depressed males. Novelty seeking, harm avoidance, reward dependence and self directedness all had significant linear relationships with the symptom variables. Cooperativeness and self transcendence had significant linear and non-linear relationships with the symptom variables, involving phobic anxiety with psychotocism or anxiety. Persistence had no significant relationship with any of the symptom predictors.

Comparison of the Males Before and After Treatment

Harm avoidance has the same model before and after treatment. It is significantly linearly related to interpersonal sensitivity. Cooperativeness is significantly related to psychotocism at both time points. At baseline anxiety and anger hostility are also important, whereas after treatment phobic anxiety is important. Self transcendence is significantly related to phobic anxiety both before and after treatment. At baseline paranoid ideation and interpersonal sensitivity are also significantly related to self transcendence. After

Model Number	Personality Variable (Dependent Variable)	Significant Linear Predictors (Parametric Model)	Significant Non-linear Predictors (Non-parametric Model)
1	Novelty Seeking	Obsessive Compulsive	
2	Harm Avoidance	Interpersonal Sensitivity	
3	Reward Dependence	Interpersonal Sensitivity	
4	Persistence	No Model	
5	Self Directedness	Depression	
6	Cooperativeness	Psychotocism	Phobic Anxiety
7	Self Transcendence	Phobic Anxiety	Anxiety

Table C.6: Best model from GAMs for the depressed males post treatment.

treatment anxiety is significantly related to self transcendence.

Novelty seeking was not significantly related to any of the symptoms variables at baseline. After treatment, however, it was significantly related to obsessive compulsive symptoms. Reward dependence related to somatisation and anxiety at baseline and to interpersonal sensitivity after treatment. Obsessive compulsive symptoms were significantly related to persistence at baseline. After treatment no symptoms variables were found to be significantly related to persistence. Self directedness related to obsessive compulsive symptoms and interpersonal sensitivity at baseline. After treatment it was only related to depression. This post treatment model is the only male model that depression plays a significant role in.

Comparison of the Males and Females

There are some similarities between the baseline models for the males and females. Harm avoidance is significantly related to interpersonal sensitivity. Self directedness is also significantly related to interpersonal sensitivity for both males and females. Cooperativeness is significantly related to anger hostility. After treatment harm avoidance is still significantly related to interpersonal sensitivity for both the males and females. Cooperativeness is now significantly related to phobic anxiety for both the males and females.

C.3 Investigation of the R^2

Further investigation of the simplest model, predicting novelty seeking linearly from anger hostility for the baseline depressed females (Model 2, Table C.3), gave an R^2 value of 0.0183. To investigate this further the relationship between anger hostility and novelty seeking was plotted using a scatter plot with the regression line shown (Figure C.3). Even though the slope is significantly different from zero, anger hostility is a poor predictor of

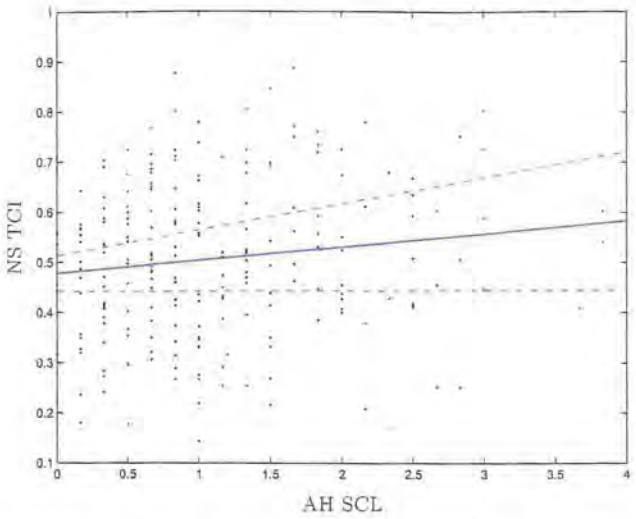


Figure C.3: Anger hostility as a predictor of novelty seeking for the depressed females at baseline. Key: —— is the regression line , with – – – 95% confidence interval.

harm avoidance. The following sections uses the R^2 and R^2_{adj} to investigate which models are reasonable predictors of the personality variables.

Table C.7 presents the results from analysing the multiple linear regression models. The F-test for overall model fit shows that all the models have good fits, however analysis of the residuals by way of the R^2_{adj} statistic shows that the symptoms are all poor predictors of personality. Only one R^2_{adj} value is above 30%, so this is the only model where the symptom variable is explaining more than 30% of the variance in the personality model. This model (Model 5, Table C.3) will be further investigated graphically in the next section. Similar analysis was conducted for the non-linear models (Table C.8). The R^2_{adj} values for three of the models is more than 30% so these models (Model 3 and Model 6, Table C.4; Model 6, Table C.6) will be further investigated, the other models are poor predictors of personality, even though there are significant relationships.

C.4 Investigation of the Models with $R^2_{adj} > 0.3$

The Female's Baseline Self Directedness

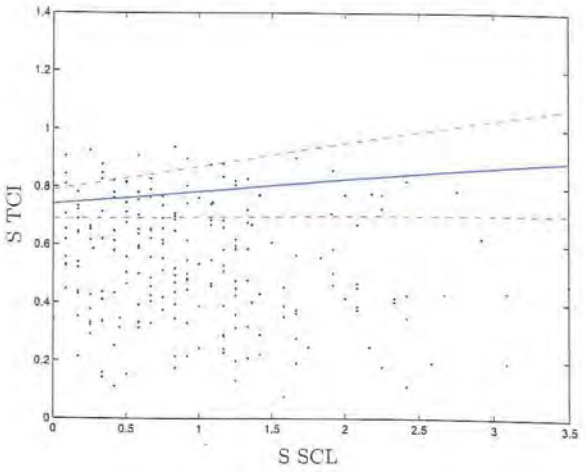
The first model analysed is between self directedness and the three symptoms of somatisation, interpersonal sensitivity and psychotocism for the depressed females at baseline (Model 6, Table C.3). Figure C.4 presents scatter grams showing the dependent variable, self directedness (TCI), versus each of the independent symptom variables, somatisation, interpersonal sensitivity and psychotocism. Somatisation appears to have the weakest relationship, interpersonal sensitivity and psychotocism show negative relationships with

Multiple Regression		F-test				R^2	R^2_{adj}
	Models	F	df_{num}	df_{den}	p-value		
Females Baseline	TCI Factor	24	4	217	< 0.0001	0.3079	0.2952
	Novelty Seeking	4.1	1	220	0.0441	0.0183	0.0138
	Harm Avoidance	39.71	2	219	< 0.0001	0.2662	0.2595
	Persistence	6.14	2	219	0.0025	0.0531	0.0445
	Self Directedness	35.41	3	218	< 0.0001	0.3276	**0.3184**
	Cooperativeness	20.62	2	219	< 0.0001	0.1585	0.1508
	Self Transcendence	11.2	2	219	< 0.0001	0.0928	0.0845
Females Post treatment	TCI Factor	39.02	1	133	< 0.0001	0.2268	0.221
	Cooperativeness	14.5	2	132	< 0.0001	0.1801	0.1677
	Self Transcendence	4.31	1	133	0.0398	0.0314	0.0241
Males Baseline	Harm Avoidance	18	1	122	< 0.0001	0.1286	0.1215
	Reward Dependence	6.76	2	121	0.0017	0.1004	0.0856
	Persistence	4.7	1	122	0.0322	0.0371	0.0292
	Self Directedness	13.48	2	121	< 0.0001	0.1822	0.1687
	Cooperativeness	10.14	3	120	< 0.0001	0.2023	0.1824
Males Post treatment	Novelty Seeking	6.33	1	65	0.0143	0.0888	0.0748
	Harm Avoidance	24.14	1	65	< 0.0001	0.2708	0.2596
	Reward Dependence	8.13	1	65	0.0058	0.1111	0.0975
	Self Directedness	24.21	1	65	< 0.0001	0.2714	0.2602

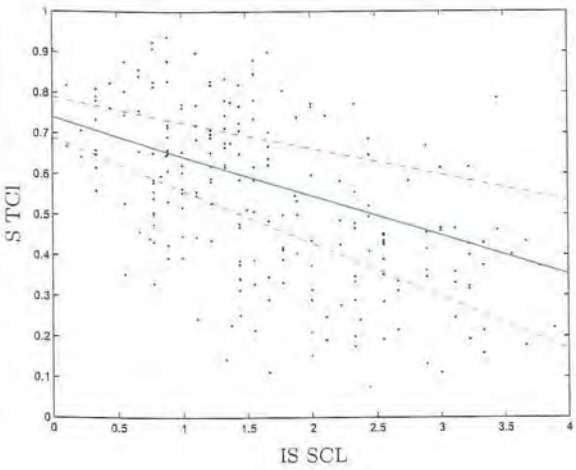
Table C.7: R^2 analysis of the multiple linear regression models.

Non-linear Models		R^2	R^2_{adj}
Females Post treatment	Novelty Seeking	0.1151	0.0948
	Harm Avoidance	0.471	**0.4505**
	Reward Dependence	0.2276	0.2159
	Persistence	0.2261	0.2083
	Self Directedness	0.3582	**0.3484**
Males Baseline	Self Transcendence	0.2796	0.2616
Males Post treatment	**Cooperativeness**	0.4043	**0.3857**
	Self Transcendence	0.3068	0.2852

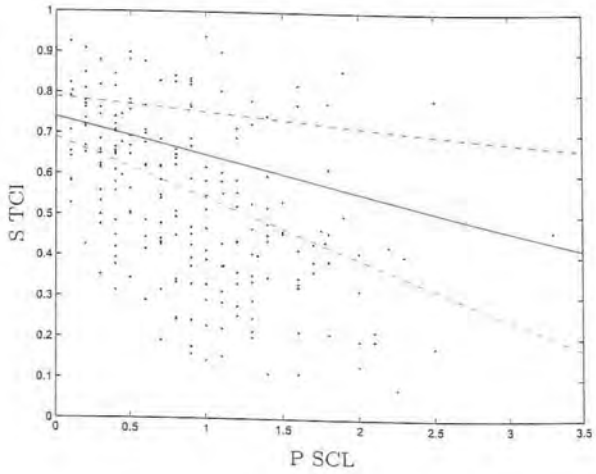
Table C.8: R^2 analysis of the semi-parametric models.



(a) Somatisation



(b) Interpersonal Sensitivity



(c) Psychotocism

Figure C.4: Symptoms as predictors of self directedness for the depressed females at baseline (Model 6, Table C.3). Key: ——— is the regression line , with – – – 95% confidence interval.

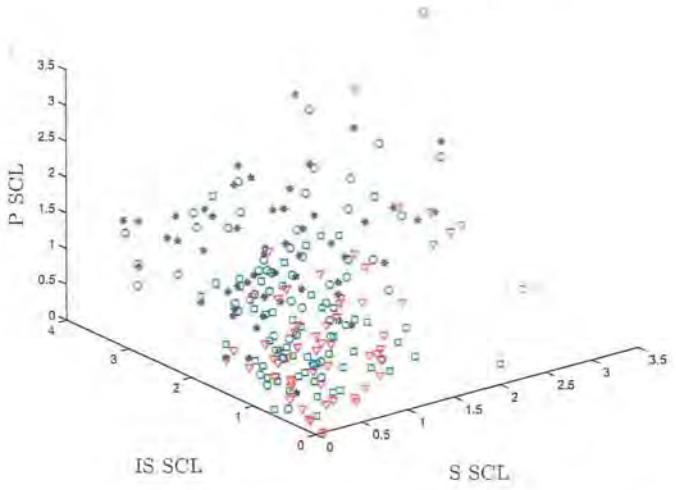
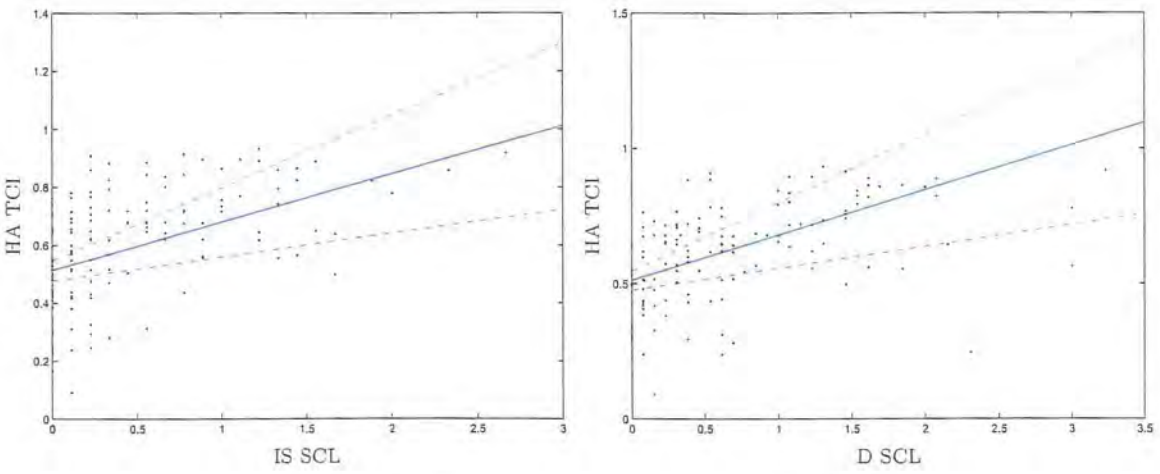


Figure C.5: The three symptom predictors of self directedness (Model 6, Table C.3) with self directedness colour coded for the depressed females at baseline. Key: $*$ are the observations below Q1, $\circ$ are the observations between Q1 and Q2, $\square$ are the observations between Q2 and Q3, $\triangledown$ are the observations greater than Q3.

self directedness. Figure C.5 presents a three dimensional plot showing the three independent variables. The dependent variable has been colour coded by quartile levels. The graph clearly shows that people with high self directedness are at the low ends of both interpersonal sensitivity and psychotocism. The graphs also show that the relationship between somatisation and self directedness is weak, even though the slope is significantly different from zero.

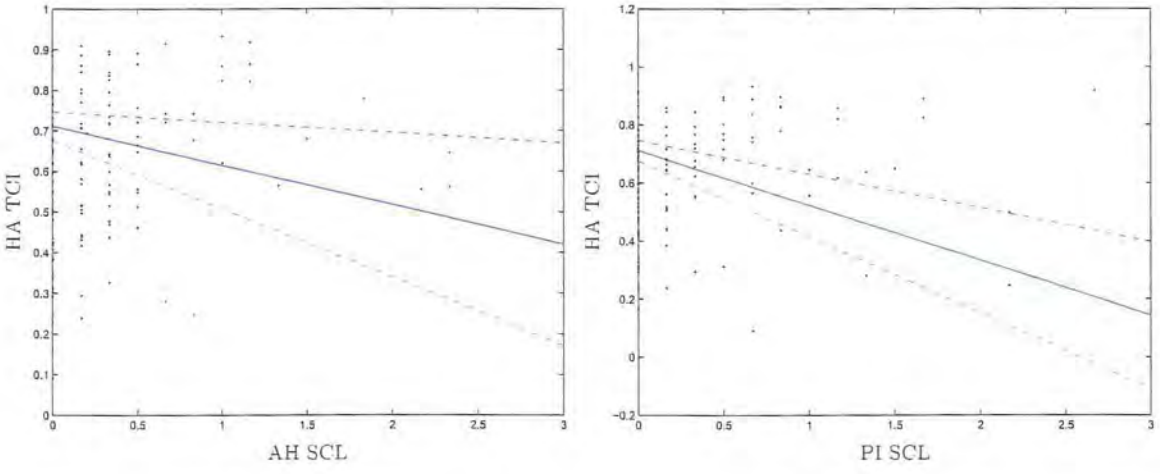
The Female's Post Treatment Harm Avoidance

The second model analysed is the first of the semi parametric models. It predicts harm avoidance scores for the depressed females post treatment, linearly from interpersonal sensitivity, depression, anger hostility and paranoid ideation, and nonlinearly from obsessive compulsive (Model 3, Table C.3). Figure C.6 presents scatter grams for each of the symptoms that are linearly related to harm avoidance. The point-wise 95% confidence interval on the gradient is large in all four cases and it is clear from the graph why the R^2_{adj} value is low. Anger hostility and paranoid ideation have very poor fits. The symptoms, whilst having a significant correlation with harm avoidance, are not good predictors of the personality trait. The partial prediction plot (Figure C.7) shows the non-linear relationship between harm avoidance and obsessive compulsive. The residuals plotted on the graph, Figure C.7, show that a few values are driving the non-linearity for high obsessive compulsive symptoms. The model was reanalysed with the extreme values removed. The new regression spline is presented in Figure C.8. The non-linear trend is still significant



(a) Interpersonal Sensitivity

(b) Depression



(c) Anger Hostility

(d) Paranoid Ideation

Figure C.6: Relationship between harm avoidance and the four linear predictor symptoms for the depressed females post treatment (Model 3, Table C.4). Key: —— is the regression line , with – – – 95% confidence interval.

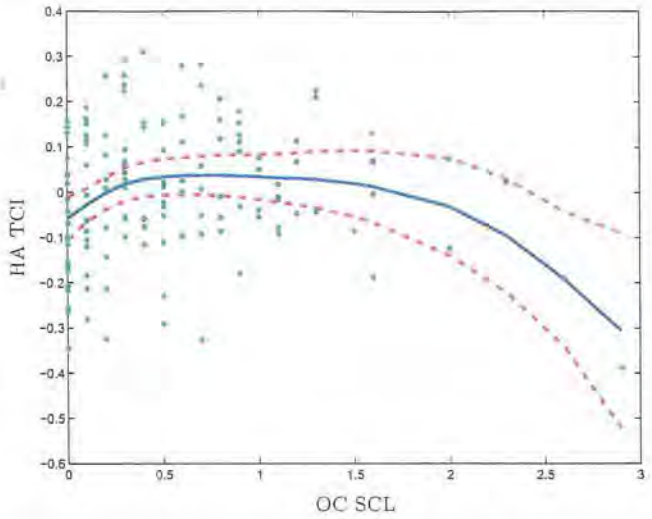


Figure C.7: Spline curve for the partial prediction of harm avoidance by obsessive compulsive symptoms for the depressed females post treatment (Model 3, Table C.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

at the 10% level. The R^2_{adj} after removal of the outliers is 0.46 compared to the original R^2_{adj} of 0.45. This model has the highest amount of variance explained. The symptom variables account for 0.46 of the variance in harm avoidance.

The Female's Post Treatment Self Directedness

Figure C.9 presents the plot of interpersonal sensitivity versus self directedness for the depressed females after treatment (Model 6, Table C.4). The regression line is significantly negative. Figure C.10 shows the partial prediction of self directedness by psychotocism, the curve is significantly non-linear and appears to be affected at high psychotocism values by outliers. As before, the model was reanalysed with removal of the outliers. The new spline curve is presented in Figure C.11, and is significantly non-linear at the 1% level. The R^2_{adj} is now 0.36 compared to 0.35 before outlier removal.

The Male's Post Treatment Cooperativeness

Figure C.12 presents the scatter plot for cooperativeness versus psychotocism for the depressed males post treatment showing the significant negative regression line. Figure C.13 presents the spline for the prediction of cooperativeness by phobic anxiety. The spline is significantly non-linear but appears to be affected by an outlier at high phobic anxiety values. The model was reanalysed with this point removed and the spline is presented in Figure C.14. The relationship between phobic anxiety and cooperativeness and is now not significantly non-linear and the R^2_{adj} value after outlier removal is 0.24 compared to

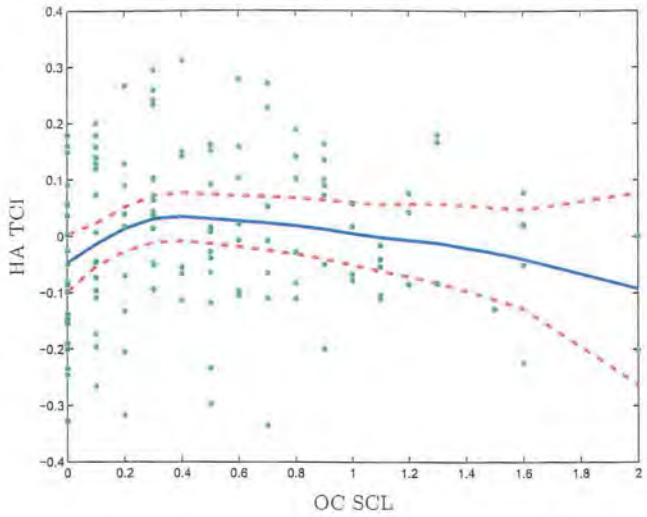


Figure C.8: Spline curve after removal of potential outliers for predicting post treatment harm avoidance in the depressed females (Model 3, Table C.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

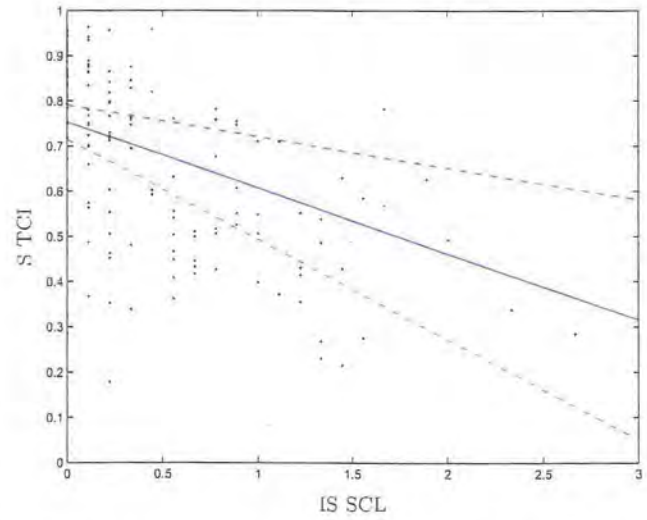


Figure C.9: Relationship between self directedness and the linear predictor symptom of interpersonal sensitivity for the depressed females post treatment (Model 6, Table C.4). Key: —— is the regression line . with – – – 95% confidence interval.

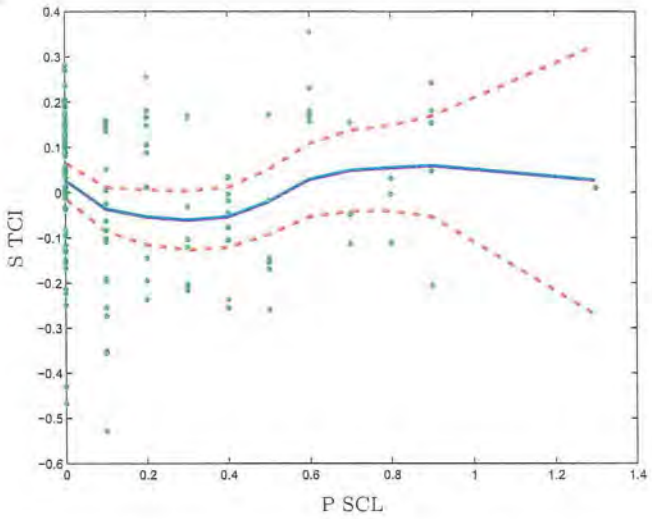


Figure C.10: Spline curve for the partial prediction of self directedness by psychotic symptoms for the depressed females post treatment (Model 6, Table C.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

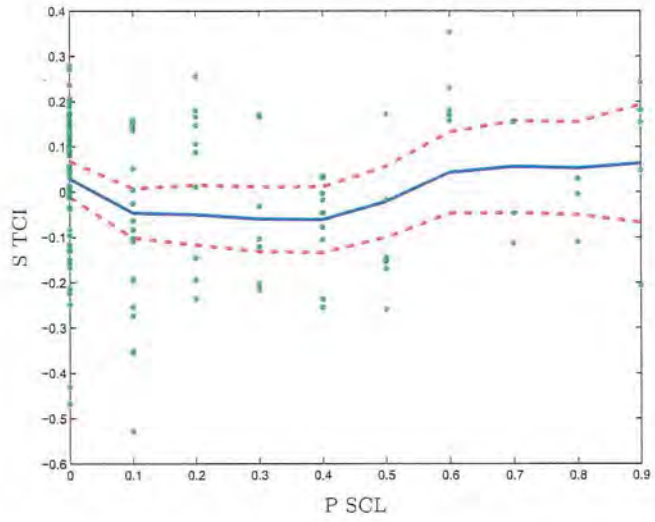


Figure C.11: Spline curve after removal of potential outliers for psychotic symptoms predicting self directedness in the post treatment females (Model 6, Table C.4). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

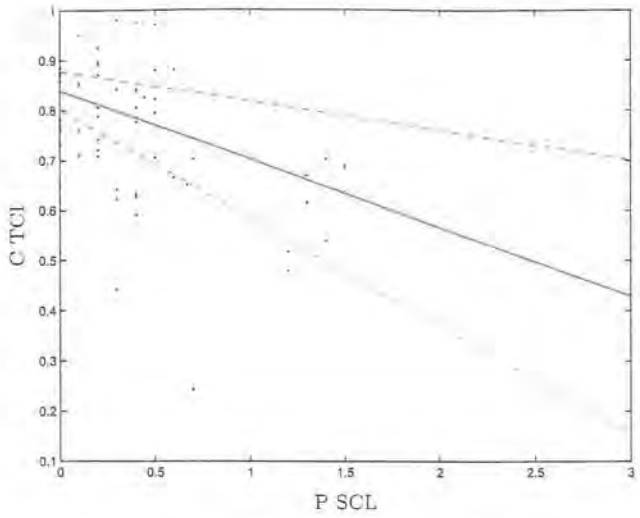


Figure C.12: Relationship between cooperativeness and the linear predictor symptom of psychotocism for the depressed males post treatment (Model 6, Table C.6). Key: —— is the regression line , with – – – 95% confidence interval.

the original 0.39 suggesting that the outlier has a strong impact on the model.

C.5 Summary

In general, symptoms are poor predictors of the personality traits even though there are significant relationships between them. The two structural models analysed, showed poor fit and insignificant loadings for the regression terms between the latent symptom and personality variables. The path analysis model is superior to multiple linear regression as it allows for the modelling of the latent structure. However, the draw back of path analysis is that it only models linear terms. The results from the path analysis show that there are no significant relationships from the symptom model to the personality model at both time points for the depressed females. The depressed males were not analysed in this manner, as at baseline, only a symptom model was available and after treatment only a personality model. The path analysis agrees with the results found in Appendix B where combined TCI and SCL models were developed. These models had few personality variables in them and when they were involved in the model, the factors did not have significant covariances with the symptom factors.

To thoroughly investigate any potential relationship from symptoms to personality, general additive models (GAMS) were used. There were a number of significant linear and non-linear relationships between personality and symptoms. Analysis of the residuals, via the R^2 and R^2_{adj} measures, revealed that whilst significant relationships existed the symptoms were poor predictors of personality.

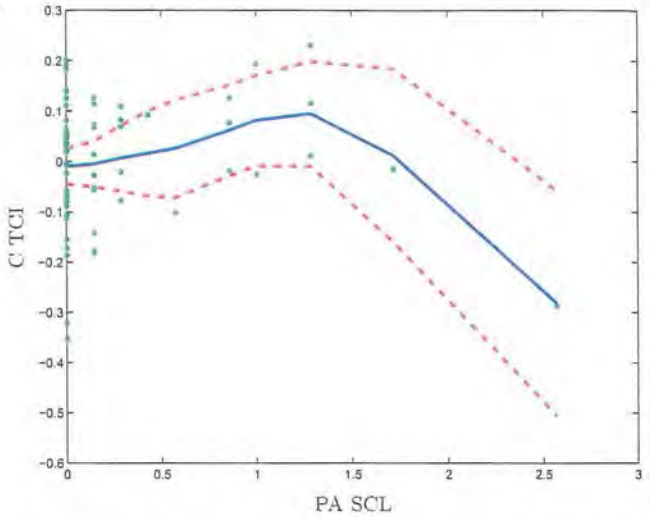


Figure C.13: Spline curve for the partial prediction of cooperativeness by the symptom of phobic anxiety for the depressed males post treatment (Model 6, Table C.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

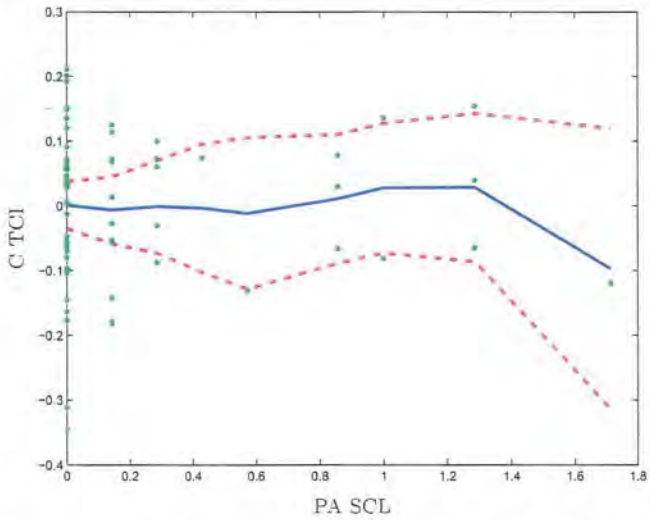


Figure C.14: Spline curve after removal of potential outliers for the prediction of cooperativeness in the post treatment males by phobic anxious symptoms (Model 6, Table C.6). Key: —— is the regression line , with – – – 95% confidence interval and ∗partial residuals.

The best model found was for the depressed females after treatment (Model 3, Table C.4). Approximately 45% of the variance in harm avoidance could be explained by interpersonal sensitivity, depression, anger hostility, paranoid ideation and obsessive compulsive symptoms. The first four symptoms were linear predictors, whilst obsessive compulsion was non-linear. Low levels of interpersonal sensitivity, depression and high levels of anger hostility, paranoid ideation and obsessive compulsive predict low levels of harm avoidance.

Two other models (Model 6, Table C.3 and Model 6, Table C.4) were able to explain approximately 30% of the variance of a particular trait, whilst the other models were all below 30%. This suggests that whilst some general trends can be established, i.e. for depressed females post treatment high interpersonal sensitivity scores relate to low self directedness scores; actual prediction of personality traits from symptoms will be poor and manifest large errors.

The significant relationships found are summarised as follows. The baseline females have the following significant relationships with the symptom variables, models with an $R^2_{adj} > 0.30$ are shown in bold.

$$F1_{TCI} = -0.22F1_{SCL} + 0.47F2_{SCL} + 0.28F3_{SCL} + 0.21F4_{SCL} - 0.87$$

$$NS_{TCI} = 0.03AH_{SCL} + 0.48$$

$$HA_{TCI} = 0.14IS_{SCL} - 0.07PI_{SCL} + 0.55$$

$$RD_{TCI} = none$$

$$P_{TCI} = -0.07IS_{SCL} + 0.05S_{SCL} + 0.54$$

$$\mathbf{S_{TCI} = 0.04S_{SCL} - 0.09PI_{SCL} - 0.10IS_{SCL} + 0.74}$$

$$C_{TCI} = -0.11PI_{SCL} + 0.07PA_{SCL} + 0.87$$

$$ST_{TCI} = 0.03OC_{SCL} + 0.05S_{SCL} + 0.20$$

The following models represent the males at baseline.

$$NS_{TCI} = none$$

$$HA_{TCI} = 0.10IS_{SCL} + 0.51$$

$$RD_{TCI} = 0.10A_{SCL} - 0.07S_{SCL} + 0.49$$

$$P_{TCI} = 0.06OC_{SCL} + 0.39$$

$$S_{TCI} = -0.06OC_{SCL} - 0.06IS_{SCL} + 0.71$$

$$C_{TCI} = -0.06AH_{SCL} - 0.08P_{SCL} + 0.05A_{SCL} + 0.85$$

$$ST_{TCI} = 0.07PI_{SCL} + f(IS_{SCL}) + f(PA_{SCL}) + 0.28$$

Models were developed for the post treatment data. The best models for the females are shown below.

$$F1_{TCI} = f(F2_{SCL})$$

$$NS_{TCI} = -0.11A_{SCL} + 0.07PI_{SCL} + f(S_{SCL}) + 0.52$$

$$\mathbf{HA_{TCI} = 0.17IS_{SCL} + 0.17D_{SCL} - 0.10AH_{SCL} - 0.19PI_{SCL} + f(OC_{SCL}) + 0.51}$$

$$RD_{TCI} = -0.07PI_{SCL} + f(S_{SCL}) + 0.76$$

$$P_{TCI} = -0.19S_{SCL} + f(PI_{SCL}) + f(PA_{SCL}) + 0.55$$

$$\mathbf{S_{TCI} = -0.15IS_{SCL} + f(P_{SCL}) + 0.75}$$

$$C_{TCI} = -0.03AH_{SCL} - 0.04PI_{SCL} + 0.88$$

$$ST_{TCI} = 0.07PI_{SCL} + 0.30$$

The post treatment models for the males are below.

$$NS_{TCI} = -0.06OC_{SCL} + 0.55$$

$$HA_{TCI} = 0.17IS_{SCL} + 0.47$$

$$RD_{TCI} = -0.07IS_{SCL} + 0.65$$

$$P_{TCI} = none$$

$$S_{TCI} = -0.14D_{SCL} + 0.73$$

$$C_{TCI} = -0.14P_{SCL} + f(PA_{SCL}) + 0.84$$

$$ST_{TCI} = -0.18PA_{SCL} + f(A_{SCL}) + 0.28$$

There appears to be more significant relationships post treatment between the symptoms and personality than at baseline. Two of the post treatment models for the females had R^2_{adj} values above 30% and only one baseline model had an R^2_{adj} above 30%, though this reduced, to below 30%, when a potential outlier was removed from the model. The baseline female models were all linear, where as the post treatment models are more complicated with non-linear terms in six of the eight models.

Interpersonal sensitivity was positively and linearly related to harm avoidance in both males and females at both time points. The models for cooperativeness are interesting. At baseline high values of anger hostility are related to low values of cooperativeness for both the males and females. After treatment anger hostility does not feature in the models, but phobic anxiety does for both males and females. For the females, paranoid ideation is negatively related to cooperativeness at both time points, and for the males psychotocism is negatively related to cooperativeness at both time points.

Interpreting the Three Models with $R^2_{adj} > 0.3$

From the female baseline models, high self directedness is predicted by low somatisation, high interpersonal sensitivity and high psychotocism (Figure C.5). For the females after treatment, two models have symptom variables that are reasonable predictors of the two personality traits. The first model has low levels of interpersonal sensitivity and depression and high levels of anger hostility and paranoid ideation predicting high levels of harm

avoidance (Figure C.6). This model also includes a non-linear term. The spline curve C.8 shows that the maximum values of harm avoidance occur for values of obsessive compulsive around 0.4, values above or below this have lower values of harm avoidance. The second model predicts high self directedness levels for low interpersonal sensitivity scores (Figure C.9) and the spline curve (Figure C.11) suggests that self directedness values decrease as psychotocism increases up to a value of about 0.4 then the self directedness values increase with increasingly psychotic symptoms.

The Depression Symptom Predicting Personality

The study involves a dataset of depressed patients so the most relevant symptom to them is depression. Depression was a predictor of personality in two models. Both of these models were for the post treatment data. In the first model depression was one of five symptoms predicting the female's harm avoidance scores. The relationship between depression and harm avoidance was positive and linear. Low levels of depression predicted low levels of harm avoidance. In the second model depression is the single predictor of self directedness and the relationship is linear and negative. Thus, low depression levels predicted high self directedness levels.

Appendix D

Anatomical Brain Maps

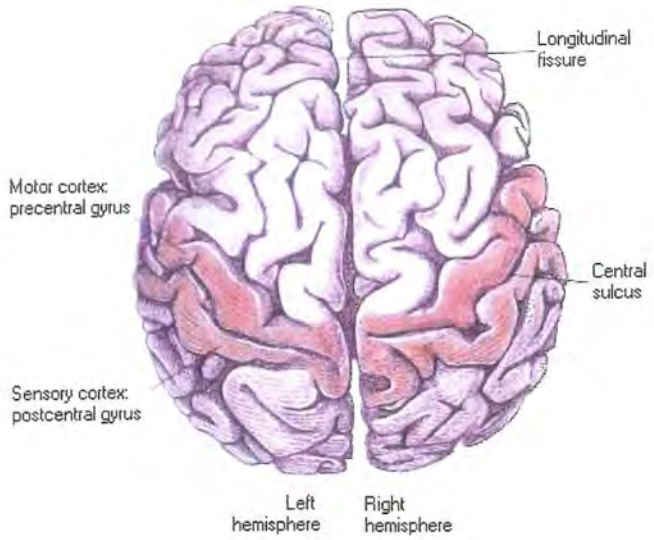


Figure D.1: The human cerebral cortex with left and right hemisphere, viewed from above (Bloom and Lazerson, 1988).

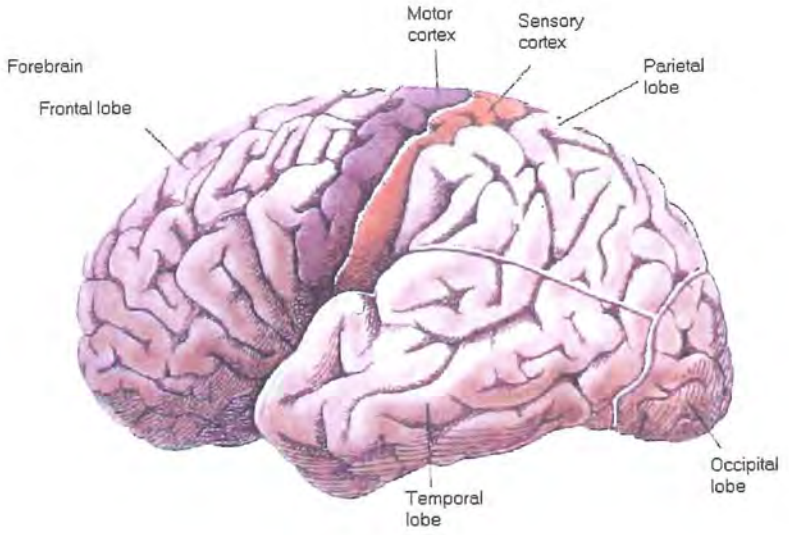


Figure D.2: Side view of one hemisphere showing the division into four lobes (Bloom and Lazerson, 1988).

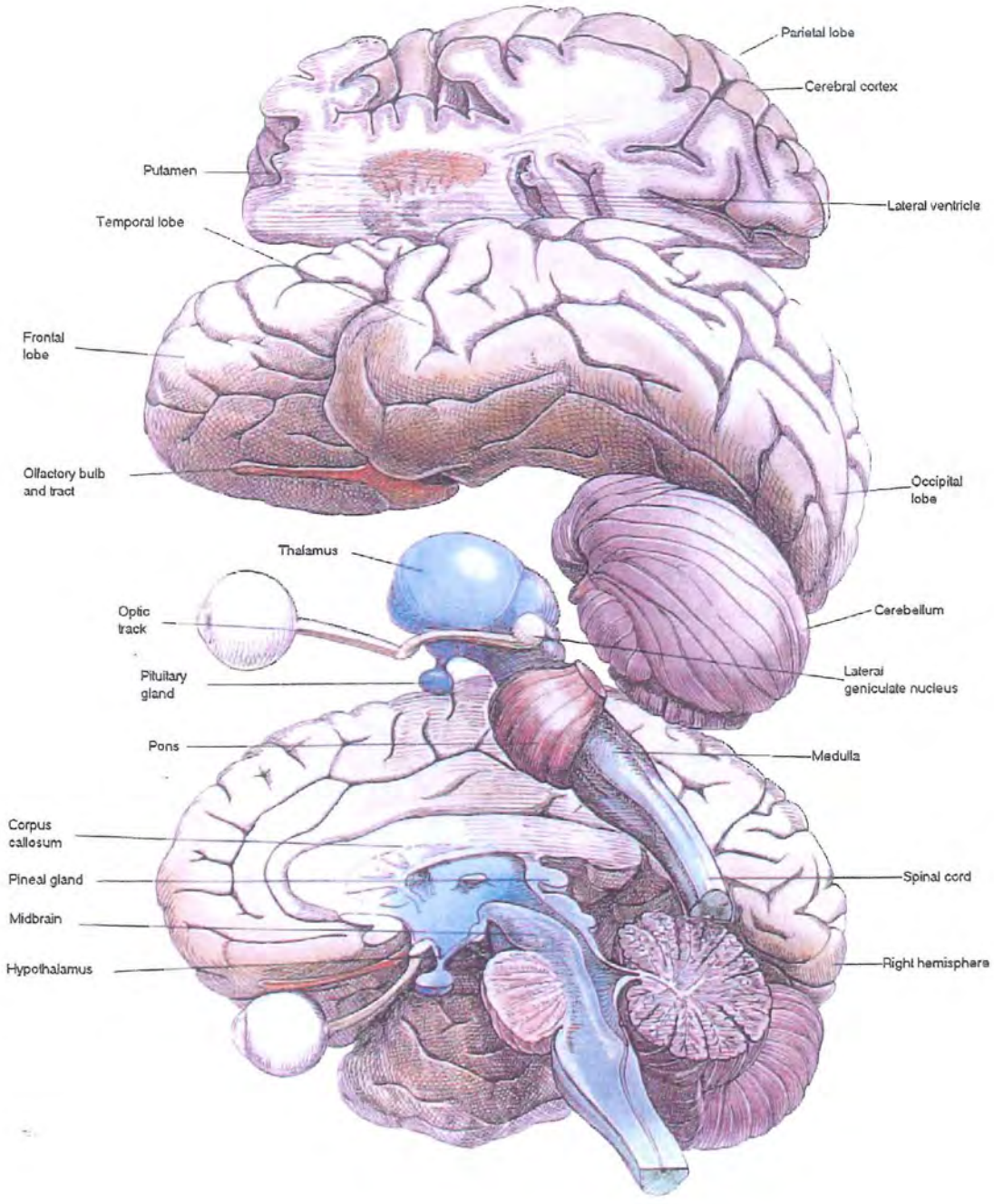


Figure D.3: A sliced and separated brain to show the major areas (Bloom and Lazerson, 1988).

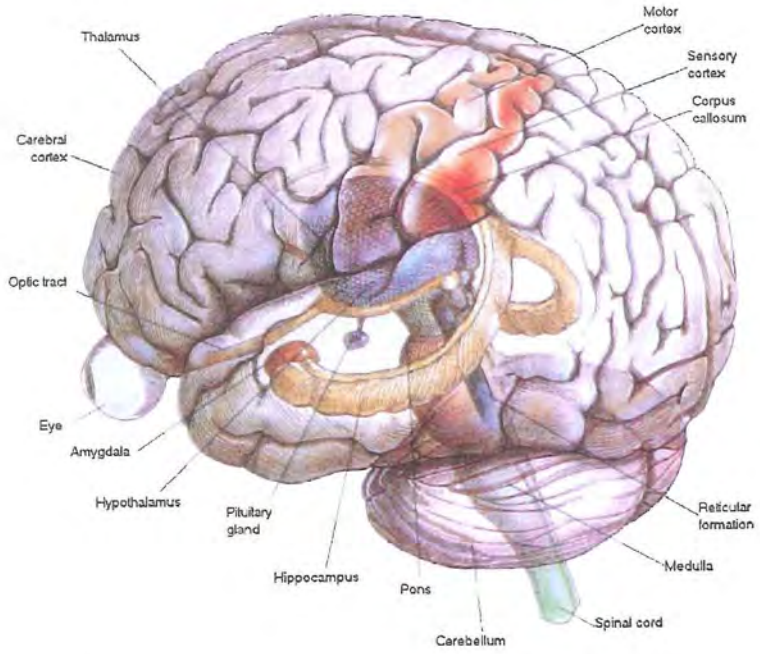


Figure D.4: The assembled brain showing the major areas (Bloom and Lazerson, 1988).

Appendix E

Information Sheet

Regional Brain Blood Flow and Personality in Healthy Males

Introduction

You are invited to take part in a project which will study the connection between personality traits and blood flow patterns in the brain. We ask you to read the information about the project that has been supplied to you, and take a few days to consider the advantages and consequences before deciding whether or not you wish to participate. You do not have to take part, and you can withdraw from the project at any time.

About the Study

Why are we doing the study?

The aim of the project is to identify the relationship between personality types and regional brain blood flow. Any relationship will help identify any key areas that are associated with differences in personality type. The study will use a gamma camera to image the distribution of a radioactive tracer. This is called Single Photon Emission Computed Tomography (SPECT). This will provide a three-dimensional image of the blood flow in the brain.

Why are we using SPECT?

We are using the SPECT method of imaging as it is the only available method that provides a functional image. Other common imaging techniques such as Computed Tomography (CT) scans and Magnetic Resonance Imaging (MRI) only provide a structural image. This tells us what the brain looks like but gives us no indication of its function or amount of blood flow in certain regions.

Selection Criteria

You will be offered a chance to participate in this project if you meet the selection criteria. We are looking for twenty healthy males aged between 20 and 50 years. You need to have no health problems including present or past head injuries, neurological diseases and psychiatric illness, as they may confuse results. You must have no family history of psychiatric illness due to the hereditary nature of these illnesses. Medication and drugs will also confuse results so you need to be clean of these for the appropriate wash out period of the particular drug. You will also be excluded from the study if you are a shift worker, radiation worker or if you have had a SPECT scan in the last two years.

How will the study be conducted?

There will be one consultation needed. This will be held at

Nuclear Medicine Department
Floor 2
Riverside Block
Christchurch Hospital

There will be an interview to obtain a medical and social history of you. Robin Turner will conduct this interview. The medical history is to obtain details of any past head injuries, neurological disease, psychiatric illness, and medication use. These are important as they may effect blood flow in unknown ways and confuse results. The social history is needed for social demographics such as age, suburb, profession which may be related to the personality types. You will also be required to fill out a personality questionnaire & a psychiatric symptom checklist. The personality questionnaire will be analysed to give us your people type. The psychiatric symptom checklist will be analysed to screen for any psychiatric disorders. You may choose not to answer individual questions if these cause you particular concern. The interview should take about 2 hours to complete. The information that you provide in this interview will remain confidential. Technologists from the Nuclear Medicine Department will then conduct the scanning.

What to expect

On arrival at the Nuclear Medicine Department you will be familiarized with the scanning equipment. You will be interviewed and the radioactive isotope will be injected into your arm. The scanning will commence 30 minutes after the injection. The scanning will involve lying still while a gamma camera rotates around your head. This will take approximately 30 minutes. This completes your involvement in the study.

Risks and Benefits

The radioactive isotope is called 99m-technetium HMPAO. This emits gamma rays of energy 141keV. This is the same sort of radiation that is used in hospital x- rays. The isotope has a half life of 6 hours, this means that after every six hours the radiation activity halves. Exposure to radiation is linked to increases in the risk of developing cancer and has a potential for the development of hereditary defects. The following table will give you an indication of the risk involved in this study.
Approximate lifetime risks of fatality from various causes in New Zealand
All types of cancer 2300 per 10 000
Motor vehicle accidents 160 per 10 000
Accidental falls 50 per 10 000
Homicide 20 per 10 000
Drowning 20 per 10 000
Fire 6 per 10 000
Our study 3.29 per 10 000
Accidental Poisoning 2 per 10 000
One year's natural background radiation 2 per 10 000

Other side effects include a few reported cases of an allergic reaction. There can be risks and discomforts in having an intravenous injection. It might cause pain and bruising.

If you participate in this study you will be helping scientists to understand the connections between personality and brain function. With better understanding psychiatrists hope to be able to provide better care for the psychiatric ill and perhaps prevent psychiatric illness. There will be no charge for participating in this study.

Participation

Your participation is entirely voluntary (your choice). You do not have to take part in this study, and if you choose not to take part this will not affect any future treatment.

If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your future health care.

Participation in this study will be stopped should any harmful effects appear or if the doctor feels it is not in the participant's best interests to continue.

General

Your GP will not be informed that you are in this study. You can obtain further information about the study by contacting Mrs Turner at the following address.
Robin Turner

Department of Mathematics and Statistics
University of Canterbury
Private Bag 4800
Christchurch
Ph. (03) 364 2987 extn 8873

If you have any queries or concerns about your rights as a participant in this study you may wish to contact a Health and Disability Services Consumer Advocate, telephone (03)377 7501

Confidentiality

No material which could personally identify you will be used in any reports on this study. You will be given a number. Your name and number will be recorded on a piece of paper and kept in a locked filling cabinet in the Department of Mathematics and Statistics at the University of Canterbury. Any other information collected will only use your number. The records will remain with the research group while the group is still conducting research into this area or for ten years. The University of Canterbury requires data to be kept for a minimum of ten years. This is so the research team can continue to extend the study. The raw data will be destroyed after ten years if the research team is no longer continuing in this field. This data, which includes the personality questionnaire and the brain image, can only be accessed by Robin Turner (principal investigator), Dr Irene Hudson (supervisor), Professor Joyce (co-supervisor), Associate Professor Butler (co-supervisor), and Dr John Turner (physician).

Results

Results will be published in International Journals. Your results will also be made available to you if you request them. Copies of the reports and your results will be available from Mrs R. Turner.

Compensation

If you suffer physical injury as a result of your participation in this trial, you may be covered by ACC. You should note, however, that eligibility for cover is not automatic.

Your claim for cover may be accepted by ACC but your entitlement to compensation will depend on a number of factors such as whether you are an earner or a non-earner. You should note that in most cases ACC provides only partial reimbursement of costs and expenses and there is no lump sum compensation payable under the current ACC legislation.

If you have suffered mental injury, there will be no ACC compensation available.

You should also be aware that if you have cover under the ACC legislation your right to sue the researchers or anyone else involved in the clinical trial is extremely limited.

If you have any questions about cover or entitlements under the ACC scheme you should contact your nearest ACC branch office for further information before you consent to participate in this trial.

This study has received ethical approval from the Canterbury Ethics Committee.

Appendix F

TCI Questionnaire

Read each statement carefully, but don't spend too much time deciding on the answer. Please answer every statement by placing the appropriate number in the box after each question, even if you are not completely sure of the answer. Use 1 for False and 2 for True. Remember there are no right or wrong answers - just describe your own personal opinions and feelings. 1=False 2=True

1. I often try new things just for fun and thrills, even if most people think it is a waste of time
2. I usually am confident that everything will go well even in situations that worry most people
3. I am often moved deeply by a fine speech or poetry
4. I often feel that I am the victim of circumstances
5. I can usually accept other people as they are, even when they are very different from me
6. I believe that miracles happen
7. I enjoy getting revenge on people who hurt me
8. Often when I am concentrating on something, I lose awareness of the passage of time
9. Often I feel that my life has little purpose or meaning
10. I like to help find a solution to problems so that everyone comes out ahead
11. I could probably accomplish more than I do, but I don't see the point in pushing myself harder than is necessary to get by
12. I often feel tense and worried in unfamiliar situations, even when others feel there is little to worry about
13. I often do things based on how I feel at the moment without thinking about how they were done in the past
14. I usually do things my own way, rather than giving in to the wishes of other people
15. I often feel so connected to the people around me that it is like there is no separation

between us

16. I generally don't like people who have different ideas from me
17. In most situations my natural responses are based on good habits that I have developed
18. I would do almost anything legal in order to become rich and famous, even if I would lose the trust of many old friends
19. I am much more reserved and controlled than most people
20. I often have to stop what I am doing because I start worrying about what might go wrong
21. I like to discuss my experiences and feelings openly with friends instead of keeping them to myself
22. I have less energy and get tired more quickly than most people
23. I am often called 'absent-minded' because I get so wrapped up in what I am doing that I lose track of everything else
24. I seldom feel free to choose what I want to do
25. I often consider another person's feelings as much as my own
26. Most of the time I would prefer to do something a little risky (like riding in a fast automobile over steep hills and sharp turns) rather than having to stay quiet and inactive for a few hours
27. I often avoid meeting strangers because I lack confidence with people I do not know
28. I like to please other people as much as I can
29. I like old 'tried and true' ways of doing things much better than trying 'new and improved' ways
30. Usually I am not able to do things according to their priority of importance to me because of lack of time
31. I often do things to help protect animals and plants from extinction
32. I often wish that I was smarter than everyone else
33. It gives me pleasure to see my enemies suffer
34. I like to be very organized and set up rules for people whenever I can
35. It is difficult for me to keep the same interests for a long time because my attention often shifts to something else
36. Repeated practice has given me good habits that are stronger than most momentary impulses or persuasion
37. I am usually so determined that I continue to work long after other people have given up
38. I am fascinated by the many things in life that cannot be scientifically explained
39. I have many bad habits that I wish I could break
40. I often wait for someone else to provide a solution to my problems

41. I often spend money until I run out of cash or get into debt from using too much credit
42. I think I will have very good luck in the future
43. I recover more slowly than most people from minor illnesses or stress
44. It wouldn't bother me to be alone all the time
45. Often I have unexpected flashes of insight or understanding while relaxing
46. I don't care very much whether other people like me or the way I do things
47. I usually try to get just what I want for myself because it is not possible to satisfy everyone anyway
48. I have no patience with people who don't accept my views
49. I don't seem to understand most people very well
50. You don't have to be dishonest to succeed in business
5 1. I sometimes feet so connected to nature that everything seems to be part of one living organism
52. In conversations I am much better as a listener than as a talker
53. I lose my temper more quickly than most people
54. When I have to meet a group of strangers, I am more shy than most people
55. I am more sentimental than most people
56. I seem to have a "sixth sense" that sometimes allows me to know what is going to happen
57. When someone hurts me in any way, I usually try to get even
58. My attitudes are determined largely by influences outside my control
59. Each day I try to take another step toward my goals
60. I often wish I was stronger than everyone else
61. I like to think about things for a long time before I make a decision
62. I am more hard-working than most people
63. I often need naps or extra rest periods because I get tired so easily
64. I like to be of service to others
65. Regardless of any temporary problem that I have to overcome, I always think it will turn out well
66. It is hard for me to enjoy spending money on myself, even when I have saved plenty of money
67. I usually stay calm and secure in situations that most people would find physically dangerous
68. I like to keep my problems to myself
69. I am often troubled by the difficulties I have dealing with others
70. I like to stay at home better than to travel or explore new places
71. I do not think it is smart to help weak people who cannot help themselves

72. I cannot have any peace of mind if I treat other people unfairly, even if they are unfair to me
73. People will usually tell me how they feel
74. I often wish I could stay young forever
75. Sometimes I get upset
76. Sometimes I have felt like I was part of something with no limits or boundaries in time or space
77. I sometimes feel. a spiritual connection to other people that I cannot explain in words
78. I try to be considerate of other people's feelings, even when they have been unfair to me in the past
79. I like it when people can do whatever they want without strict rules and regulations
80. I would probably stay relaxed and outgoing when meeting a group of strangers, even if I were told they are unfriendly
81. Usually I am more worried than most people that something might go wrong in the future
82. I usually think about all the facts in detail before I make a decision.
83. I feel it is more important to be sympathetic and understanding of other people than to be practical and tough-minded
84. I often feel a strong sense of unity with all the things around me
85. I often wish I had special powers like Superman
86. Other people control me too much
87. I like to share what I have learned with other people
88. Religious experiences have helped me to understand the real purpose of my life
89. I often learn a lot from people
90. Repeated practice has allowed me to become good at many things that help me to be successful
91. I am usually able to get other people to believe me, even when I know that what I am saying is exaggerated or untrue
92. I need much extra rest, support, or reassurance to recover from minor illnesses or stress
93. I know there are principles for living that no one can violate without suffering in the long run
94. I don't want to be richer than everyone else
95. I would gladly risk my own life to make the world a better place
96. Even after thinking about something a long time, I have learned to trust my feelings more than my logical reasons
97. Sometimes I have felt my life was being directed by a spiritual force greater than any human being

98. I usually enjoy being mean to anyone who has been mean to me
99. I have a reputation as someone who is very practical and does not act on emotion
100. It is easy for me to organize my thoughts while talking to someone
101. I haven't got as far as I'd like to in life because of the kind of person I am
102. I am strongly moved by sentimental appeals (like when asked to help crippled children)
103. I usually push myself harder than most people do because I want to do as well as I possibly can
104. I have so many faults that I don't like myself very much
105. I have too little time to look for long-term solutions for my problems
106. I often cannot deal with problems because I just don't know what to do
107. I often wish I could stop the passage of time
108. I hate to make decisions based only on my first impressions
109. I prefer spending money rather than saving it
110. I can usually do a good job of stretching the truth to tell a funnier story or to play a joke on someone
111. Occasionally I talk about people behind their backs
112. If I am embarrassed or humiliated, I get over it very quickly
113. It is extremely difficult for me to adjust to changes in my usual way of doing things because I get so tense, tired, or worried
114. I usually demand very good practical reasons before I am willing to change my old ways of doing things
115. I need a lot of help from other people to train me to have good habits
116. I think that extra-sensory perception (ESP like telepathy or precognition) is really possible
117. I would like to have warm and close friends with me most of the time
118. A nuclear war may not be such a bad idea
119. I nearly always stay relaxed and carefree, even when nearly everyone else is fearful
120. I find sad songs and movies pretty boring
121. Circumstances often force me to do things against my will
122. It is hard for me to tolerate people who are different from me
123. I think that most things that are called miracles are just chance
124. I would rather be kind than get revenge when someone hurts me
125. I often become so fascinated with what I'm doing that I get lost in the moment – like I'm detached from time and place
126. I do not think I have a real sense of purpose for my life
127. I try to cooperate with others as much as possible
128. I am satisfied with my accomplishments, and have little desire to do better

129. I often feel tense and worried in unfamiliar situations, even when others feel there is no danger at all
130. I often follow my instincts, hunches, or intuition without thinking through all the details
131. Other people often think that I am too independent because I won't do what they want
132. I often feel a strong spiritual or emotional connection with all the people around me
133. It is usually easy for me to like those people who have different values from me
134. Other people often seem bothered by the things I do or say
135. Good habits have become 'second nature' to me – they are automatic and spontaneous actions nearly all the time
136. I don't mind the fact that other people often know more than I do about something
137. I usually try to imagine myself "in other people's shoes", so I can really understand them
138. Principles like fairness and honesty have little role in some aspects of my life
139. I am better at saving money than most people
140. I have never told a lie
141. Even when most people feel it is not important, I often insist on things being done in a strict and orderly way
142. I feel very confident and sure of myself in almost all social situations
143. My friends find it hard to know my feelings because I seldom tell them about my private thoughts
144. I hate to change the way I do things, even if many people tell me there is a new and better way to do it
145. I think it is unwise to believe in things that cannot be explained scientifically
146. I like to imagine my enemies suffering
147. I am more energetic and tire less quickly than most people
148. I like to pay close attention to details in everything I do
149. I often stop what I am doing because I get worried, even when my friends tell me everything will go well
150. I often wish I were more powerful than everyone else
151. I usually am free to choose what I will do
152. Often I become so involved in what I am doing that I forget where I am for a while
153. Members of a team rarely get their fair share
154. Most of the time I would prefer to do something risky (like hang-gliding or parachute jumping), rather than having to stay quiet and inactive for a few hours
155. Because I so often spend too much money on impulse, it is hard for me to save money, even for special plans like a vacation

156. I don't go out of my way to please other people
157. I am not shy with strangers at all
158. I often give in to the wishes of friends
159. I spend most of my time doing things that seem necessary but not really important to me
160. I don't think that religious or ethical principles about what is right and wrong should have much influence in business decisions
161. I often try to put aside my own judgments so that I can better understand what other people are experiencing
162. Many of my habits make it hard for me to accomplish worthwhile goals
163. I have made real personal sacrifices in order to make the world a better place – like trying to prevent war, poverty, and injustice
164. I never worry about terrible things that might happen in the future
165. I almost never get so excited that I lose control of myself
166. I often give up a job if it takes much longer than I thought it would
167. I prefer to start conversations, rather than waiting for others to talk to me
168. Most of the time I quickly forgive anyone who does me wrong
169. My actions are determined largely by influences outside my control
170 The way I behave often gets me into trouble on the job, at school or at home
171. I prefer to wait for someone else to take the lead in getting things done
172. I usually respect the opinions of others
173. I have had experiences that made my role in life so clear to me that I felt very excited and happy
174. It is fun for me to buy things for myself
175. I believe that I have experienced extra-sensory perception myself
176. I believe that my brain is not working property
177. My behaviour is strongly guided by certain goals that I have set for my life
178. It is usually foolish to promote the success of other people
179. I often wish I could live forever
180. I usually like to stay cool and detached from other people
181. I am more likely to cry at a sad movie than most people
182. I recover more quickly than most people from minor illnesses or stress
183. I often break rules and regulations when I think I can get away with it
184. I need much more practice in developing good habits before I will be able to trust myself in many tempting situations
185. I wish other people didn't talk as much as they do
186. Everyone should be treated with dignity and respect, even if they seem to be unimportant or bad

187. I like to make quick decisions so I can get on with what has to be done
188. I usually have good luck in whatever I try to do
189. I am usually confident that I can easily do things that most people would consider dangerous (such as driving an automobile fast on a wet or icy road
190. I am bothered by the kind of person I am
191. I like to explore new ways to do things
192. I enjoy saving money more than spending it on entertainment or thrills
193. Individual rights are more important than the needs of any group
194. I have had personal experiences in which I felt in contact with a divine and wonderful spiritual power
195. I have had moments of great joy in which I suddenly had a clear, deep feeling of oneness with all that exists
196. Good habits make it easier for me to do things the way I want
197. Most people seem more resourceful than I am
198. Other people and conditions are often to blame for my problems
199. It gives me great pleasure to help others, even if they have treated me badly
200. I often feel like I am a part of the spiritual force on which all life depends
201. Even when I am with friends, I prefer not to "open up" very much
202. I usually can stay 'on the go" all day without having to push myself
203. I nearly always think about all the facts in detail before I make a decision, even when other people demand a quick decision
204. I am not very good at talking my way out of trouble when I am caught doing something wrong
205. I am more of a perfectionist than most people
206. Whether something is right or wrong is just a matter of opinion
207. I think my natural responses now are usually consistent with my principles and long-term goals
208. I believe that all life depends on some spiritual order or power that cannot be completely explained
209. I think I would stay confident and relaxed when meeting strangers, even if I were told they are angry at me
210. People find it easy to come to me for help, sympathy, and warm understanding
211. I am slower than most people to get excited about new ideas and activities
212. I have trouble telling a lie, even when it is meant to spare someone else's feelings
213 There are some people I don't like
214. I don't want to be more admired than everyone else
215. Often when I look at an ordinary thing, something wonderful happens – I get the feeling that I am seeing it fresh for the first time

216. Most people I know look out only for themselves, no matter who else gets hurt
217. I usually feet tense and worried when I have to do something new and unfamiliar
218. I often push myself to the point of exhaustion or try to do more than I really can
219. Some people think I am too stingy or tight with my money
220. Reports of mystical experiences are probably just wishful thinking
221. My will power is too weak to overcome very strong temptations, even if I know I will suffer as a consequence
222. I hate to see anyone suffer
223. I know what I want to do in my life
224. I regularly take time to consider whether what I am doing is right or wrong
225 Things often go wrong for me unless I am very careful
226. If I am feeling upset, I usually feel better around friends than when left alone
227. I don't think it is possible for one person to share feelings with someone else who hasn't had the same experiences
228. It often seems to other people like I am in another world because I am so completely unaware of things going on around me
229. I wish I were better looking than everyone else
230. I have lied a lot on this questionnaire
231. I usually stay away from social situations where I would have to meet strangers, even if I am assured that they will be friendly
232. I love the blooming of flowers in the spring as much as seeing an old friend again
233. I usually look at a difficult situation as a challenge or opportunity
234. People involved with me have to learn how to do things my way
235. Dishonesty only causes problems if you get caught
236. I usually feel much more confident and energetic than most people, even after minor illnesses or stress
237. I like to read everything when I am asked to sign any papers
238. When nothing new is happening, I usually start looking for something that is thrilling or exciting

Appendix G

SCL Questionnaire

Below is a list of problems and complaints that people sometimes have. Please read each one carefully and then place a cross (X) in the box that best describes how much that problem has bothered or distressed you during the past week including today. Mark only one box for each problem and do not skip any items.

HOW MUCH WERE YOU BOTHERED BY

1. Headaches
2. Nervousness or shakiness inside
3. Unwanted thoughts, words or ideas that won't leave your mind
4. Faintness or dizziness
5. Loss of sexual interest or pleasure
6. Feeling critical of others
7. The idea that someone else can control your thoughts
8. Feeling others are to blame for most of your troubles
9. Trouble remembering things
10. Worried about sloppiness or carelessness
11. Feeling easily annoyed or irritated
12. Pains in heart or chest
13. Feeling afraid in open spaces or on the streets
14. Feeling low in energy or slowed down
15. Thoughts of ending your life
16. Hearing voices that other people do not hear
17. Trembling
18. Feeling that most people cannot be trusted
19. Poor appetite
20. Crying easily
21. Feeling shy or uneasy with the opposite sex

22. Feeling of being trapped or caught
23. Suddenly scared for no reason
24. Temper outbursts that you could not control
25. Feeling afraid to go out of your house alone
26. Blaming yourself for things
27. Pains in lower back
28. Feeling blocked in getting things done
29. Feeling lonely
30. Feeling blue
31. Worrying too much about things
32. Feeling no interest in things
33. Feeling tearful
34. Your feelings being easily hurt
35. Other people being aware of your private thoughts
36. Feeling others do not understand you or are unsympathetic
37. Feeling that people are unfriendly or dislike you
38. Having to do things very slowly to ensure correctness
39. Heart pounding or racing
40. Nausea or upset stomach
41. Feeling inferior to others
42. Soreness of your muscles
43. Feeling that you are watched or talked about by others
44. Trouble falling asleep
45. Having to check and double-check what you do
46. Difficulty making decisions
47. Feeling afraid to travel on buses, subways or trains
48. Trouble getting your breath
49. Hot or cold spells
50. Having to avoid certain things, places or activities because they frighten you
51. Your mind going blank
52. Numbness or tingling in parts of your body
53. A lump in your throat
54. Feeling hopeless about the future
55. Trouble concentrating
56. Feeling weak in parts of your body
57. Feeling tense or keyed up
58. Heavy feelings in your arms or legs
59. Thoughts of death or dying

60. Overeating
61. Feeling uneasy when people are watching or talking about you
62. Having thoughts that are not your own
63. Having urges to beat, injure or harm someone
64. Awakening in the early morning
65. Having to repeat the same actions such as touching, counting, washing
66. Sleep that is restless or disturbed
67. Having urges to break or smash things
68. Having ideas or beliefs that others do not share
69. Feeling very self-conscious with others
70. Feeling uneasy in crowds such as shopping or at a movie
71. Feeling everything is an effort
72. Spells of terror or panic
73. Feeling uncomfortable about eating or drinking in public
74. Getting into frequent arguments
75. Feeling nervous when you are left alone
76. Others not giving you proper credit for your achievements
77. Feeling lonely when you are with people
78. Feeling so restless you couldn't sit still
79. Feelings or worthlessness
8O. Feeling that familiar things are strange or unreal
81. Shouting or throwing things
82. Feeling afraid you will faint in public
83. Feeling that people will take advantage of you if you let them
84. Having thoughts about sex that bother you a lot
85. The idea that you should be punished for your sins
86. Feeling pushed to get things done
87. The idea that something serious is wrong with your body
88. Never feeling close to another person
89. Feelings of guilt
90. The idea that something is wrong with your mind

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